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NATURE, SCOPE AND SIGNIFICANCE OF ANISEIKONIA

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Aniseikonia is divided, for classification, into two types, normal and abnormal, or *anomalous,* as some ophthalmologists prefer to call it. Both types have long been recognized but not fully studied. As a result of Professor Ames's intensive researches, order is emerging from the chaos.

In the literature, normal aniseikonia is dealt with under the terms "disparity of the retinal images," which is simply the Latin for inequality of the retinal images, and "incongruity of the images," which means that the image of one eye does not exactly fit that of the other. Why does it not fit? Simply because the images are different in size and shape. Aniseikonia is merely a Greek word meaning images that are not equal, that differ in size and shape. It is probable that the term aniseikonia will more and more be used for *abnormal* differences in size and shape, i. e., the anomalous incongruities, since a single word for such differences is convenient. Writers from the Dartmouth Eye Institute have used the word in this restricted sense.

What I wish to emphasize is that fundamentally the basis of binocular space perception is found in the normal differences in size and shape of the retinal images of objects in space by which one knows where they are—much about their relative size and shape and position—since these determine the size, shape and position of the retinal images. Normal aniseikonia, or retinal disparity, is the basis of stereoscopic depth perception, in which the two eyes, because of their lateral separation of 60 to 70 mm., get two different views of an object or of a group of objects (a field of view). The visual processes make use of this inequality, these differences between the two ocular images, for better perception of the form and the spatial relations of the objects. These differences in the

The opinions expressed in this paper are my own and do not always coincide with those of my colleagues at the Dartmouth Eye Institute.

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images are differences in size and shape, i. e. aniseikonia; for example, the relative position of points in the two images in a stereoscopic picture (fig. 1).

There are other normal forms of aniseikonia. One is the effect of nearness on the relative size of the image of an object on the nasal as compared with the temporal part of the retina, the significance of which Professor Ames[1] has pointed out and permits me to quote

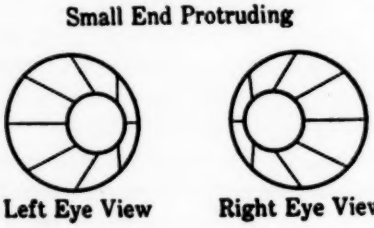

Fig. 1.—Binocular perspective. When this figure is viewed in a stereoscope, the distance of the small circle from the large circle on the right side is much larger as seen by the right eye than as seen by the left eye. The effect of this aniseikonia is to make the small circle appear much nearer to the observer. It is not the two long lines that are fused with each other, but the long one seen by one eye is fused with the short one seen by one eye is fused with the short one seen by the other eye.

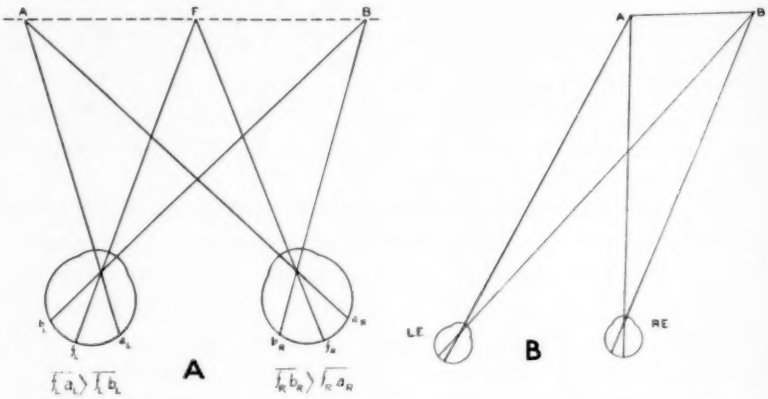

Fig. 2.—Normal forms of aniseikonia. *A*, greater size of the nasal portions of the retinal images of near objects as compared with the temporal portions. The image of *AF* is larger on the left retina, where it is nasal, than it is on the right retina, where it is temporal. *B* difference in dioptric images produced by asymmetric convergence. The object *AB* is to the right of the midline and therefore nearer the right eye than the left. Hence the retinal image of the right eye is larger than that of the left eye.

1. Ames, A., Jr.: Unpublished data.

(fig. 2 *A*). He has shown why a flat prism base in will cause the nasal image of a nearby object in one eye to be larger than the temporal image of the same object in the other eye, thus explaining why prisms base in make objects appear nearer, although they cause divergence, which is associated with greater distance.

Another form of normal aniseikonia is seen in asymmetric convergence. Here the dioptric image of the object on the retina of the nearer eye (the abducted eye) will be larger than the image on the retina of the eye which is farther away (the adducted eye; fig. 2 *B*). The difference in size may easily be 5 to 10 per cent or even more, but it is "cheerfully borne" (Ludvigh). The reason is that ever since binocular vision began, the eyes have had to see that way, and they not only tolerate it, they take advantage of it to locate with greater precision the position in space of the object with reference to the observer. This is not surprising, since the difference in size is due to the position of the object.[2]

In looking at nearby objects to right, to left, up or down—in any direction away from the primary position—since the two eyes are separated an appreciable distance, there will be differences in the images which depend on the geometric relations; e. g., looking straight up will have a different effect than looking up to the right or up to the left and, of course, a very different effect from looking to the right or to the left or down. The amount and the kind of difference are the clues by which one knows in what direction the object is located. Formerly it was thought that knowledge of the direction of gaze was derived from proprioceptive sensations, but that is certainly a mistake. It is by the characteristic differences in size and in shape of the ocular images (i. e., by the aniseikonia, the normal incongruities) that one locates objects in one's environment. Unless one grasps this conception, asymmetric convergence is an unsolved riddle.

Anomalous aniseikonia has been classified as: (1) meridional, axis 90 degrees; (2) meridional, axis 180 degrees; (3) over all, same magnification in all meridians; (4) cyclo type—(*a*) due to oblique cylinders and (*b*) due to cyclophoria—and (5) asymmetric type. Each type produces characteristic effects on spatial localization (fig. 3).

A rectangle of pasteboard about 30 by 50 cm. is held vertically 2 or 3 meters in front of the observer in the midline in his frontal plane. The images of this rectangle as seen by the two eyes are equal.

1. Rotate the rectangle around its vertical axis, and the image as seen by the right eye is different from that seen by the left.

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2. "Since there is no reason to limit its powers, we will assume that the central nervous system is able to get every scrap of information out of the message." (Adrian, E. D.: The Basis of Sensation, New York, W. W. Norton & Company, 1928.)

2. Rotate the rectangle around its horizontal axis.

3. Move the rectangle in its same plane to the right or to the left or up or down.

4. Bend the rectangle so that it is convex or concave toward the observer.

With each of these procedures the normal aniseikonia gives the clue to the observer as to the position of the rectangle. With the rectangle in its original position, suitable lenses could be placed before the eyes to make the rectangle appear in any one of these different positions (artificial aniseikonia).

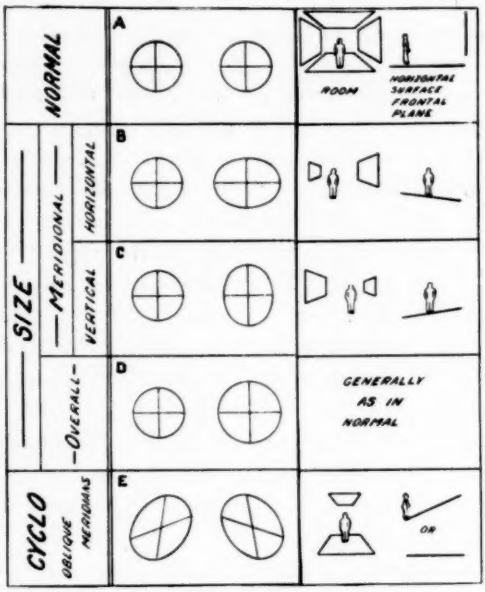

Fig. 3.—Examples of some effects of four types of aniseikonia on localization in a leaf room. *A*, normal vision. *B*, right image larger in the horizontal meridian. This makes the right wall appear larger and farther away than the left wall. The observer seems to be moved to the left. The ground slopes down to the right. *C*, right image larger in the vertical meridian. This makes the left wall larger and farther away. The observer seems to be moved to the right. The ground slopes down to the left. *D*, because *B* and *C* neutralize each other, the effects mentioned do not occur. Other effects (not shown) appear on the space eikonometer and make the diagnosis definite. *E*, oblique meridional size differences (in this case the axes converge upward). This type makes the ground seem either to slope up away from the observer, (compare the effect of slanting lines viewed in a stereoscope) or to be level but lower: the observer seems to be moved up—he feels taller.

HOW ABNORMAL ANISEIKONIA IS DEALT WITH BY THE VISUAL PROCESSES

By analogy with refractive errors and defects of motility, it might be expected that some compensatory mechanism would correct the difference in size of the images.

There is no evidence of the existence of such a corrective mechanism, much less any knowledge of how it might operate. It must be concluded that the difference is dealt with in some entirely different way. Nature's favorite way of dealing with a difficult or an intolerable situation is by suppression, or inhibition, as the physiologists prefer to call it. For example, in certain defects of motility, when the compensatory mechanism fails, causing diplopia, the situation is met by suppression of the vision of one eye. This may result in amblyopia of one eye from persistent inhibition (ex anopsia), or it may result in an inhibition so nicely adjusted that when either eye fixates, the vision of the other eye is suppressed temporarily (alternating strabismus). Moreover, the suppression may affect only a limited part of the retina, allowing it to escape the unwanted effect but preserving its activity for other situations.

Suppression is by no means limited to the visual processes. It is a generally utilized device, for example in hearing and other sensory activities; in fact, civilization and etiquette depend on inhibition of instinctive impulses.

To understand how inhibition is made use of in dealing with abnormal aniseikonia, consider what abnormal aniseikonia does and what conflicts and frustrations it may excite. The binocular clues, though in many cases the most perfect means of spatial localization, are not the only ones. Very important are the monocular clues based on perspective, overlapping, shadows, knowledge of size and of form and previous experience (fig. 4).

Thus in a given situation one receives information from monocular clues, as well as from binocular clues, and what one makes of it is not determined solely by the physical stimuli but, although primarily excited by them, is modified by some mechanism at a higher level. "The image awakens unconscious reproductions or memories of past experiences which modify it so that the ultimate perception does not correspond with the immediate stimulus but conforms with the background provided by experience."[3] For example, a tilted circle gives a retinal image which is an ellipse, but if it is known to be a ring, it gives the impression of a circle in a particular position. One's impressions of the size of a well known object and of its shape do not depend solely on the size and the form of its retinal image. The phenomena of "color constancy" and

3. Duke-Elder, W. S.: Text-Book of Ophthalmology, St. Louis, C. V. Mosby Company, 1933, vol. 1, p. 1019.

"memory colors" and the effect of different conditions of light intensity and quality on color perceptions are good examples of how the visual processes put two and two and two and two together and make seven or eight or nine, as the conditions seem to warrant. The pattern that emerges must have meaning, must make sense, must conform to experience.

This is exactly what takes place in the locating of objects in space. There are clues from monocular vision, clues from binocular vision (if affected by abnormal aniseikonia they may be false or misleading clues), clues from memory, clues from past experiences and clues from other sensory sources. It is in the presence of such clues that the "interplay of the psychic processes" [4] produces a consistent, coherent, harmonious

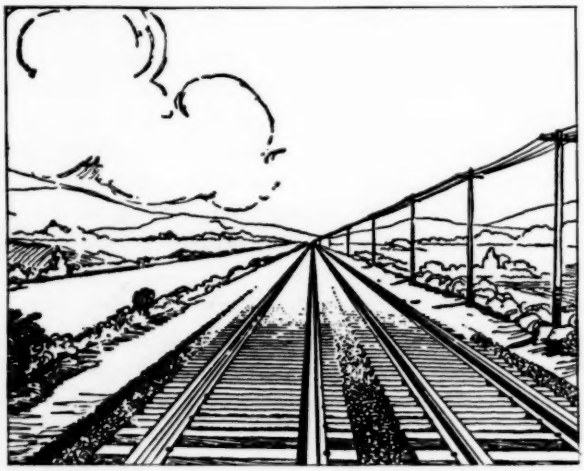

Fig. 4.—Monocular perspective. When this figure is looked at even with only one eye, there are many strong clues as to distance.

result, a perception that makes sense, that satisfies one's comprehension. To quote Duke-Elder: [5]

> It is impossible to experience a pure isolated sensation. Even the simplest and most elementary perceptual process comprises a synthesis of many types of different impressions; and to add to the complexity, the synthesized product is moulded upon and influenced by a substratum of past experience and inherited dispositions, to assess which is beyond our knowledge, while its nature is continually undergoing alterations impressed upon it by the higher centers, more especially with regard to the distribution of the psychological factors of attention and interest.

4. von Kries, J.: Helmholtz's Treatise on Physiological Optics, translated from the third German edition, edited by J. P. C. Southall, Ithaca, N. Y., The Optical Society of America, 1925, vol. 3, p. 532.

5. Duke-Elder,[3] p. 1013.

In interpreting a field of view, the visual processes throw into the discard (i. e., suppress) such clues as do not contribute to the consistent, coherent, harmonious result. One must picture to oneself a conflict going on between different factors. When correcting clues are scanty, faulty clues tend to predominate. Habit greatly facilitates the correct interpretation. Thus a person with abnormal aniseikonia (natural or induced by size lenses) will under certain conditions show no apparent defect in his spatial localization (though he may show premature fatigue from the struggle and frustration, which of course are entirely subconscious). Under other conditions (absence of correcting clues, including lack of previous experience with the environment), his orientation is faulty and confusing.

I have dwelt at some length on the psychologic aspect of the subject because it is the key to an understanding of certain questions that are sure to arise in one's mind when one gives thought to the subject: 1. Why is it that if abnormal aniseikonia is such a factor in spatial localization so few persons are aware of or show disturbed orientation? 2. How is it that aniseikonia causes symptoms of eyestrain?

1. Persons who for experimental purposes continuously wear size lenses to produce an artificial abnormal aniseikonia soon acquire a certain degree of adaptation to them (Dr. Burian has shown this and permits me to quote him). However, as soon as such a person finds himself in an environment where monocular clues and clues from memory are meager or absent, he manifests the signs and symptoms of abnormal spatial localization, no matter how long he has worn the size lenses or how fully he seems to be adapted. All this will be set forth with a wealth of detail and full experimental demonstration by Dr. Burian. In the same way, a person with natural abnormal aniseikonia (i. e., not induced artificially by wearing size lenses) shows a pronounced degree of adaptation in his daily life. However, when such a subject is placed in an environment devoid of clues from sources other than binocular vision, such as monocular clues, especially rectilinear clues and clues from knowledge of the environment, memory and past experience, he always shows that the underlying misleading clues are there. For example, a test with the horopter or with the leaf room would show their presence.

2. The question, "How is it that aniseikonia causes the symptoms of eyestrain?" is not easily answered; in fact, the answers to the questions of how errors of refraction and of motility cause the symptoms of eyestrain are far from satisfactory. The identity of the symptoms shows that they must have something in common. It has been commonly assumed that the symptoms of heterophoria are due to fatigue of a weak muscle. There are two objections to this assumption. The first is that the

symptoms are not such as would be likely to result from fatigue of a muscle; the second is that the external ocular muscles are so strong that they are not likely to become fatigued and there is no satisfactory evidence that they do. In myasthenia gravis the muscles show weakness and great fatigability, but the symptoms are not those of eyestrain. The evidence that the symptoms of eyestrain in hypermetropia with astigmatism are due to fatigue of the ciliary muscle is also unsatisfactory. In the case of abnormal aniseikonia, there is no known muscular mechanism for compensating for it, and so one is forced to seek elsewhere.

Aniseikonia interferes with the smooth functioning of the visual apparatus. Poor illumination, reading poor print or reading on a train or other moving vehicle embarrass the visual apparatus and so throw added burdens on it. These factors compel greater efforts on the part of the adjusting mechanisms to secure improved seeing, thus causing eyestrain. Similarly, aniseikonia throws an added burden on the visual apparatus by interfering with its smooth functioning.

It is an important fact that the eyes do not in ordinary easy activities make the most perfect adjustments of which they are capable. Focusing is not as exact as the eyes are capable of. On the contrary, the eyes take advantage of their depth of focus and when looking at distant objects may be focused anywhere within 0.25 to 0.50 D. of infinity. When looking at a near object, for example, a book, they often find it near enough for practical purposes to focus 0.50 D. beyond the object, or the book.

The fixation apparatus does not fixate each word, much less each letter, as the eyes skip across the page in ordinary easy reading. But when a difficult passage, perhaps poorly printed, in a foreign language or dimly lighted, is encountered, then the mechanism for fixation has to perform a much more exacting task. Similarly, the fusion apparatus demands adjustment of the visual axes of the two eyes so that images fall, not on exactly corresponding points, but only on corresponding areas. However, when the demand arises, when the presence of difficulties calls for greater efforts to secure more perfect seeing, then the eyes are whipped up to more accurate focusing, fixation and fusion, thus taxing the adjusting mechanism far more than when the visual apparatus is functioning smoothly and easily, well within its capacity.

In the case of aniseikonia there is another obvious factor to be reckoned with. The adjustments call for inhibition, for selections among conflicting clues, for dealing with frustrations—a series of adjustments on a higher level than those which deal with focusing and fixation. These are cortical, psychic and mental, though mostly subconscious. Psychiatrists are keen about the strains due to frustrations. Thus on this higher level there are adequate causes for symptoms of eyestrain.

I submit, as further food for thought, that the precise similarity of symptoms of eyestrain from the various causes (refractive errors,

heterophoria, poor lighting, aniseikonia) shows that these symptoms are traceable to fundamental causes common to all. On the one hand, aniseikonia makes demands not only on the psychic adjustments but on the adjusting mechanisms of accommodation, fixation and fusion; and, on the other hand, errors of refraction and phoria make demands not alone on the lower mechanisms but on the higher psychic activities of inhibition, mental adjustments and frustrations. The visual function is so complex but so integrated that all these factors I have mentioned work together in producing eyestrain.

The subject of space perception, although it has an importance in the theory of vision which is hard to exaggerate, is not generally taught to ophthalmologists and therefore is not known or understood by them. The best books on the subject are in German and in the case of Tschermak, perhaps the most important writer in the field, written in a difficult style. To appreciate the significance and importance of aniseikonia is impossible for those who have but a sketchy knowledge of the subject of space perception and the physiology of vision. It is a most hopeful augury that Professor Ames has mastered many of the problems of this difficult field and has in preparation an exposition of the subject.

Dartmouth Eye Institute.

ABSTRACT OF DISCUSSION

DR. ERNEST A. W. SHEPPARD, Washington, D. C.: Dr. Lancaster has defined, classified and presented the subject logically, simply and briefly.

The following classification has helped me to visualize this difficult and involved subject:

1. Physiologic: That which Dr. Lancaster has described as normal.

2. Inherent: That which exists with emmetropia or isometropia. This type might be considered as anatomic, essential or inherent.

3. Induced: (*a*) that induced by the correction of anisometropia or antimetropia; B (*b*) that induced by changes in base curve or thickness or by distance of the lens from the eye.

The importance of the induced type in everyday refraction is demonstrated by the following cases:

CASE 1.—J. R. P., a man aged 65, was examined because of presbyopic symptoms. He was emmetropic and needed a + 2.50 D. sphere for reading. The ocular examination otherwise gave essentially normal results. His prescription was ground in ultex A and neutralized correctly. He complained that while wearing his glasses he would drive his car too far to the left. The right lens was ground on a —8.00 D. base curve and the left on a —6.00 D. base curve. This difference in the base curves introduced aniseikonia or added to inherent aniseikonia sufficiently to disturb his perception of spatial relationships.

CASE 2.—C. H., a man aged 73, for whom a cataract in each eye had been successfully removed, saw clearly with each eye but complained that objects seen with the left eye were larger and that binocular vision was not comfortable. The findings were as follows:

Right eye: + 6.50 D. sph. ⌒ + 1.75 D. cyl., axis 165, vision 20/20 —4; left eye: + 8.00 D. sph. ⌒ + 1.00 D. cyl., axis 45, vision 20/20 —1.

Right eye: 9.75 mm. from the closed lid; left eye: 11.50 mm. from the closed lid.

Right eye: front curves, + 10.50 D. and + 12.25 D.; left eye: front curves, + 12.00 D. and + 13.00 D.

Right eye: center thickness, 23 points; left eye: center thickness, 24 points (5 points equal 1 mm.).

These four factors contributed to the larger image of the left eye. This case illustrates a successful surgical result but a successful refractive result only after the factors causing aniseikonia had been corrected. For further consideration of these four factors it is obvious that transposition of a prescription by the optician may change the effective dioptric power and the power and size magnifications. The following case illustrates this fact:

CASE 3.—K. F., a girl aged 15, had had repeated refractions but still had asthenopia. She was wearing orthogon lenses with the following correction:

Right eye: — 3.25 D. sph. ⌒ + 3.75 D. cyl., axis 95, on a —10.12 D. base curve.

Left eye: —2.50 D. sph. ⌒ + 2.75 D. cyl., axis 80, on a —9.12 D. base curve.

After a careful manifest and cycloplegic refraction the correction was changed to:

Right eye: — 4.25 D. cyl., axis 5

Left eye: — 3.75 D. cyl., axis 170

The optician was directed to grind the lenses on + 1.25 D. outside curves, with the cylinders inside. The patient was comfortable with this correction ground in this way.

There is no question of the place and of the importance of aniseikonia. Without its careful consideration refraction is not the meticulous examination it should be. Professor Ames's exposition of the subject is awaited with pleasure.

DR. ERIC LILJENCRANTZ, Washington, D. C.: My interest in aniseikonia lies in its possible importance for aviators. It is tautological to state that space perception is of critical importance in the handling of aircraft and that any data which add to existing knowledge concerning space perception are likely to be of great importance indeed.

It is also tautological to state that views with regard to selection and training of aviators must be of highly pragmatic nature and that the luxury of academic speculation in making practical decisions regarding fitness for flight cannot be allowed. This does not mean that theory is to be ignored in aviation, but it does mean that research must be pushed

to meaningful and practical conclusions. Fortunately, there has been an opportunity for just this to be done with respect to aniseikonia and naval aviation.

Thanks to Dr. Ames and his group, it was possible to examine, at the Naval Air Station, Pensacola, Fla., a large group of naval aviation cadets and experienced graduate naval aviators for aniseikonia and to compare the laboratory findings with actual performance in the handling of aircraft. The experiment was planned to answer the following questions:

1. What is the incidence of aniseikonia among naval aviation cadets selected in accordance with present naval standards? (Note: Naval standards require 20/20 vision in each eye without correction and allow little tolerance for correction with a cycloplegic or for errors in phoria.)

2. What is the effect of aniseikonia on performance in aviation?

3. What remedial steps appear to be indicated with respect to selection, corrective measures, training and the like?

I believe that the experimental data secured at Pensacola make it possible to give reliable answers to all these questions.

Dr. Robert H. Peckham, who directed the study at Pensacola, is to discuss the data. I shall anticipate his remarks by raising the following questions for further research:

1. Does such aniseikonia as is found among naval aviation cadets fail to impair their flight performance (*a*) because of its trivial degree, (*b*) because of compensatory mechanisms or (*c*) because binocular cues are of relatively little importance in the handling of aircraft?

2. Is the absence of subjective complaints attributable to aniseikonia among naval aviation cadets due (*a*) to the trivial degrees in which it occurs or (*b*) to the fact that aviators as a group show significantly fewer neurotic traits than do less highly selected groups?

Dr. Robert H. Peckham, Pensacola, Fla.: In order to discuss the experiments performed at Pensacola, I must point out that what I say will express my own ideas and not necessarily those of the Navy.

At the time I was doing the work, I was employed at Dartmouth College, as a civilian. The experiments were all concerned with what Dr. Lancaster has so accurately called normal aniseikonia, and my fellow workers and I were little concerned with anomalous aniseikona and were not concerned with the pathologic conditions requiring the use of aniseikonic correction.

We examined 252 aviation cadets and instructors with the instruments available and found, first, that the eikonometer, which two years ago was the standard instrument for measuring aniseikonia, revealed no aniseikonia of a great amount. Ninety-two per cent of the subjects showed less than 0.5 per cent aniseikonia on the eikonometer, and 8 per cent showed more than that. Three per cent showed more than 0.75 per cent, and 1 subject showed more than 1 per cent —1.25 per cent.

Thus, the first thing we noticed was that aniseikonia as shown by the eikonometer was not present in the carefully selected men at Pensacola.

In addition to the work with the eikonometer, we made a series of studies on the space perception of cadets. The first study concerned the response of cadets to a frontal plane set before them and aniseikonic

lenses placed so that the plane would be distorted. Their reports on the distortion were the criteria.

The second study concerned the so-called leaf room, which is a room approximately 10 feet (305 cm.) square covered with leaves. The subject stood so that he was in the center of the front area of this room. Questions were asked of him concerning the squareness of the room, which was accurately square, and aniseikonic lenses were placed before his eyes so that the room would be distorted.

His responses to the distortion were the criteria of his passing or failing in this test. As Dr. Liljencrantz has said, there are certain criteria of performance in this experiment. Men pass, or fail to learn to fly. Those men who fail to learn to fly fail for many reasons, among which may be aniseikonia or may be failure to respond correctly to visual space orientation. That will be only one of the reasons.

Consequently, the use of aniseikonic measurements will be masked in the consideration of passing and failing men on the basis of aniseikonia.

Thirty-three failures were carefully examined on the apparatus available, and 57 men known to be of excellent flight ability were examined, among the total. These two groups showed no differences in the relative amounts of aniseikonia as found on the eikonometer. Therefore, as far as the eikonometer was concerned, no differences could be shown between men who learned to fly well and men who could not learn to fly.

On the space perception apparatus used, there was a significant difference between these two groups. The nature of that difference was such that statistically we could say that there were 98 chances in 100 that there was a difference between the two groups on the basis of their space perception. Therefore, it is a good bet to say that binocular space perception is intimately concerned, in some cases, with flying.

Now to take the questions that Dr. Liljencrantz has raised. First, as to the incidence of aniseikonia among naval aviation cadets: Aniseikonia as found by the eikonometer was not present in a significant amount. It remains to be proved that space disorientation similar to that discovered in aniseikonic disorientation is a reliable means of discriminating between men, but there is no doubt that men showing space disorientation do have trouble in flying.

The third question suggested that remedial steps be taken. This question must be considered in the light of the two natures of the aniseikonia measured. No immediate use is to be found in measurements with the eikonometer. There is, undoubtedly, considerable work to be done with the problems of space perception, and it is possible that a careful measurement of performance and a separation of faults with respect to binocular space disorientation will be useful in the selection of aviation candidates or, at least, in their training.

If, for example, a student shows errors in phoria whose nature was predicted by his space disorientation, his instruction could be changed quickly to prevent the development of dangerous aviation habits.

As to the questions concerning further research that Dr. Liljencrantz has brought up: First, does the aniseikonia found fail to impair flight performance? If aniseikonia as measured by the eikonometer is the criterion, there is no proof that it demonstrates poor performance. If

the aniseikonia found is that associated with poor space perception, or, conversely, if poor space perception is a measure of aniseikonia, it does impair the flight performance to a statistically reliable degree.

The question of the importance of binocular cues in flying is not yet answered. One always hears of Wiley Post, who flew around the world, and then, with the same eyes, bashed into a mountain. I can see no statistically reliable evidence that binocular cues are either necessary or not necessary. They are probably of assistance if they are present and useful.

The absence of subjective complaints attributable to aniseikonia again raises the problem of the nature of the aniseikonia being measured. There was no aniseikonia as shown on the eikonometer. There was a spatial disorientation which might be aniseikonic. This was found in 33 per cent of the men examined. Thirty-three per cent of the 250 men showed spatial disorientation of moderate amounts, but amounts that might be aniseikonic.

One must say, therefore, that the failure to find subjective complaints may have been due to the significantly fewer neurotic traits in the highly selected cadets. It may be that aniseikonia as measured by the eikonometer is an index of the neurotic condition of the subject.

It requires more than mathematical analysis and the reasonableness of argument to show that binocular disorientation is dependent on aniseikonia. Nevertheless, there are sufficient evidence and proof in Dr. Lancaster's paper to indicate that experiments must be carried out in a statistical manner to show the relation between space disorientation and aniseikonia.

DR. WALTER B. LANCASTER, Hanover, N. H.: I want to thank Lieutenant Peckham for his discussion of this subject. I think he must have made it clear, first, that failure to be a good flier depends on a multiplicity of causes, some ocular and many not ocular. Therefore a statistical analysis of one cause cannot be expected to show a 100 per cent effect. Nevertheless, it is significant that the statistics he compiled after completing the experiments showed 98 chances in 100 that the distorted perception of space is a factor in poor flying.

I think it must be kept clearly in mind that in a comparatively new subject a great many uncertainties and problems are bound to arise. When a piece of research reaches the stage where there are no problems, it has reached the end of its usefulness.

The significant thing to me in the study of aniseikonia is that one after another of the problems that were puzzling are being solved, that the criticisms justly raised are, in many cases, though not in all, being answered.

This, I think, is a fair record for any new and important research.

EYE DOMINANCE

ITS NATURE AND TREATMENT

IRA S. WILE, M.D.
NEW YORK

To see is to perceive with the eye. Vision is more than seeing, as it contains the idea of knowing. Hence many can see without vision and many have visions without seeing.

Optical acuity does not represent the visual habit, and its measure registers only the value of each eye as an optical instrumennt. The formula for visual acuity does not indicate total visual efficiency. Even with clear monocular images, binocular efficiency may be impaired, as it involves their fusion into a single image. Thus visual acuity depends not only on the dioptric apparatus but on the retina, the optic nerve paths and the central nervous mechanism. One may further state that vision is a function of the total organization of the individual, one phase of which inheres in the mechanics of the optical apparatus, another in the cerebral cortex and still another in the personality organization.

Body-mind unity too frequently is ignored by ophthalmologists. The term "word blindness" as used by Kussmaul and the phrase "congenital word blindness" employed by Hinshelwood are based on the assumption that sight, intelligence and speech are intact in the presence of an inability to grasp the meaning of a text. These terms today are considered as representing a phase of dyslexia. Whether or not one accepts Hinshelwood's concept of "mind blindness" or Orton's theory of strephosymbolia incidental to a lack of unilateral cerebral dominance, the fact remains that despite complete optical adequacy many persons are incapable of acquiring a normal reading ability. The deficiency, usually considered an educational problem, has been ignored by ophthalmologists because they fail to regard such a deficiency as medical unless it is related to anatomic defects, such as myopia, congenital cataract or hemianopia.

Educational inadequacies are highly significant medically, not merely as symptoms of organic cerebral disorder or functional visual disorder but because reading, for example, as a basic factor in education, profoundly affects personality in terms of relative achievement. The emotional consequences of educational inadequacy become cumulative as failure in school penalizes growth of personality. Behavior disorders

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may occur as a result of a sense of inadequacy or inferiority or because of the development of various fears and anxieties that increase neurotic trends.

The eye is only one instrument in learning. The brain is more significant than the emmetropic eye. The fundamental factors in learning involve basic functions of the eye and the ear, the association of symbolic percepts in memory, the derivation of meaning by comprehension of the nature of symbols and the total cerebral organization, with, however, the final factor inherent in the total organization of the personality. The capacity of fixating and reflecting form is exceedingly important mechanically, but the organization of form into visual memories is an internal process.

Functionally, there is an image in each eye, but the synthesis is of central and not of peripheral origin, as is thoroughly understood in the solving of the problems of strabismus. Binocular vision is sensed as occurring as though there were a central cyclopean eye. A monocular person is forced to depend on images supplied by his eye, and there is but one imaged form to be centrally organized. Nonetheless, a monocular person may have difficulty if the existent eye happens not to be his dominant eye.

In binocular vision two different optical impressions call for unification. If,by reason of injury of the naturally dominant eye, a new dominance is forced on the patient, his problem is more than that of doubtful visual acuity or the development of heteropsia. The extent to which a complete fusion of forms or identification of the image occurs is related not merely to some fusion point but to the total organization. In stereoscopic vision there should be complete harmony of the total organization in reading, but this facility for vision or learning may be handicapped by toxic or hysterical amblyopia, nystagmus, tics, fatigue or anemia. There may be no distinction between the measurable visual acuity of the artist and the nonartist, but the perceptions received are dependent on more than the mere acute appreciation of form, and the differences lie in personality and in cortical organization. The eye thus is an organ of vision whose stimulus is light but whose response is derived from cortical activity and subjective factors. The cortical processes are essential in spatial localization and may be disturbed by various experiences which produce asthenopia, interfere with the occipital cortex or give rise to amaurosis, amblyopia or aphasia, as well as simple illusions or complex hallucinations.

A significant element in efficient vision is that termed eye dominance. In interpreting this, I may repeat some facts which are well known. With the eyes at rest, parallel light rays have their fusion point at infinity, while essential accommodation is necessary for vision at the near point. Such accommodation involves the position of the eye, the

lens, the span of the eye, the condition of the cornea and other optical elements of each eye but bears a direct relation to the dominant eye.

Eye dominance represents a residuum of unilateral organization of the organism. It represents phylogenetically a condition among animals whose eyes are widely separated, acting independently, often with great mobility. Many animals have a unilateral organization whereby the eye, the hand, the foot and probably the ear of the same side work as a unity, functioning in relation to the general symmetry of bodily function. This unity is manifest in man, whose muscular control of one side is localized in the cortex of the opposite side. It is further supported by the position of the cortical center for language, oral and written, in one half of the brain.

In the development of stereoscopic vision, it is essential that the two eyes focus on the same object. Contrary to current belief, their doing so does not depend on mutual efforts of all the ocular muscles but is attained when one eye stops to rest on the object and fixates it. This sighting is due to the action of muscles innervated from the cortex of the opposite side. The parallelism of the other eye in muscular function requires it likewise to stop and focus on the object fixated by the dominant eye. This act is often seen in the accommodation of the eye in near vision. The eye which fixates the object for stabilizing the direct and binocular vision is the sighting eye, i. e., the dominant eye. The ocular dominance is not determined by any known structure or refractive error or by an organic defect except, of course, in the presence of blindness, cataract of one eye or some irremediable heteropsia or other functional condition which makes it imperative for the subject to depend entirely on the unimpaired eye.

Psychologic tests, such as that of Kohs, clearly demonstrate that under certain conditions, despite emmetropic eyes, there are definite reversals of form which produce confusion. These disturbances in vision are likewise manifest in the making and reading of reversed figures and letters, the development of mirror writing and mirror reading. Some children are so confused in their own minds concerning right and left that they are unable to locate right or left save in terms of their own postural sense. While it is known that a lateral imbalance is related to ocular discomfort and faulty ocular control, and consideration is given to heterophoria, there is less recognition that the expression of lateral imbalance may be due to a difficulty lying elsewhere than in the eyes, such as nervous instability and lack of reciprocal innervation. It is recognized that imbalance will destroy clear images and conduce to confusion, but it is not always appreciated that it may be related to problems growing out of eye dominance.

This confusion suggests that the seeing eye as a recording optical instrument must be differentiated from vision as a whole. The entire

person visualizes. This explains hysterical amblyopia despite emmetropic eyes. As the entire person is organized functionally, vision is related to the level of intelligence, the dominant handedness, the body chemistry, the organic states and the psychic processes. These may even affect acuity of sight.

As optical instruments, the eyes are effective for learning, especially in the acquisition of forms, which process includes the perception, retention and interpretation of figures, letters and words. Congenital word blindness (Hinshelwood) has been found to be related to the functional balance of the eye, the hand and the intelligence in general. The impressions of form are definitely affected by eye dominance, and the ability to reproduce the form images varies with the freedom of unilateral cerebral dominance in affecting the efforts of eye-hand coordination. The existence of central vision and emmetropia does not affect the loci in space of the forms envisioned, though it may condition the distinctness of the images. If the eye dominance is in any way uncertain, confused images result, with a consequent handicap to learning.

The relation of handedness to eyedness being omitted for the present, it is clearly important to diagnose eye dominance. Various methods have been employed for the purpose:

1. The simplest is to ask the subject to look through a cylinder at a distant object. The eye preferred for this act usually is the sighting eye.

2. Another method involves a circular aperture ½ inch (1.3 cm.) in diameter in an 8 by 10 inch card. The person being examined is asked to hold the card at arm's length, grasping a lower corner with each hand, and to look through the aperture with both eyes at a pencil or object held in the hands of the examiner or at a distinct object across the room. It is simple to observe the eye which is actually sighting through the opening. Frequently a child will actually place the card directly in front of one eye. It is also useful to ask the patient to look through the opening at an object held about 3 or 4 feet (90 or 122 cm.) from it. His doing so enables the examiner, looking through the opening, actually to see the dominant eye fixed on the object.

3. Another method, at times supportive, is to offer a stick to be sighted as though it were a gun. The dominant eye is evident.

4. Among the instruments sometimes employed are Parson's manoptoscope and Bett's telebinocular, but these are not essential for general examination.

5. Occasionally supplementary information is noted from the fact that the child finds it more difficult to close at command the lid of the dominant eye than that of the nondominant eye. The difference may be so pronounced that the child finds it necessary to hold down the lid of the dominant eye when asked to look at an object only through the nondominant eye.

Tests for eye dominance should be given with and without glasses, if they are worn. If physical optical errors are corrected properly, eye dominance is unaltered. Dominance does not depend on refractive errors, as Gould alleged. The visual confusion that hampers learning is of central rather than of peripheral origin. Hence many victims of dyslexia are not helped by corrections following routine examinations of the eye.

From the clinical standpoint it is essential to differentiate handedness as well as eye dominance. Dominant handedness represents the hand of inherent power due to some hereditary factor in neural organization, and dominant left handedness is a mendelian recessive trait. There are, however, many persons whose native handedness has been changed, in some temporarily because of injury, in others partially for some process, such as writing, and in still others by total conversion, so that the hands no longer operate in harmony with the dominant eye. The resultant altered function of the hands is complicated variously in terms of the primary relationship between handedness and eye dominance. Unilateral cortical function is the common rule, but contralateral neural pathways tend to create muscular and visual confusion. In a right-handed, right-eyed world, left handedness and left eyedness constitute hazards. This fact is suggested in the term gaucherie.

Educational complications are fewer if a unilateral status is native and maintained, while complications are prone to arise according to the degree of distortion of the native neural organization and the extent of the deviation from the socially basic right-eyed, right-handed status. Some confusion in learning is suffered by many persons who are left-eyed and left-handed, but their difficulties are tremendously increased if the original left-handed status becomes converted by training to right handedness. Even the presence of right eyedness does not assure learning efficiency if native left handedness becomes changed to right handedness. This fact will be given further consideration later.

The problems of eye dominance cannot be dissociated from the total organization of the personality, including the level of intelligence and the degree of emotional stability. All children with the left eye dominant do not have difficulties, any more than all syphilitic persons manifest dementia paralytica. Nor do all children converted from sinistrality to dextrality, or vice versa, acquire educational disabilities. Management of the problems of eye and hand dominance and relationship cannot ignore other factors inherent in the total personality. While the primary symptoms of dysfunction are most commonly reflected in lack of achievement, such as dyslexia or inadequacy in spelling, arithmetic or speech, a considerable group of persons evidence many dysgenic reactions as a result of a catastrophic incompetency. For this reason the diagnostic import of eye dominance is especially significant.

The prognostic factor likewise should be appreciated by the ophthalmologist concerned with the part that eye dominance plays in general well-being. As will be noted later, the symptoms are diverse, varying from a severity synonymous with the idea of word blindness to mere minor reversals of figures. The earlier the pathologic phase is recognized the better for adequate treatment. The mental level of the child is one item to be considered in the prognosis for educability; the emotional stability is another. The function of the eye in the development of an idea and the idea of the eye as a sensory instrument both serve the whole personality, even though the eye is a definite part of this structure. The psychologic element of individual life is a by-product of total organization, not merely of the mechanical factors involved in seeing but of the physiologic elements, represented in part by eye dominance. Learning

TABLE 1.—*Age Distribution*

Age	Number	Per Cent
4- 4:11	1	2
5- 5:11	2	4
6- 6:11	6	12
7- 7:11	12	24
8- 8:11	11	22
9- 9:11	11	22
10-10:11	4	8
11-11:11	2	4
12 and over	1	2
Total	50	100

symbols is one expression of the total organization of the personality for cultural adaptation.

For clinical study I selected from the children's health class at Mount Sinai Hospital 50 children with dyslexia. No monocular or deaf children and none handicapped by an intelligence quotient below 80 were included. The group was thus a selected one, which served better than random cases to illustrate the symptoms related to eye dominance. Being a group free from mental deficiency, it offered an excellent sampling for outlining of management of the eye dominance factor. The group contained 39 boys and 11 girls brought to the clinic because of some maladjustment in behavior or personality. Their age distribution, based on the ages at which they were brought to the clinic, is shown in table 1.

The behavior problems were of many kinds, including educational failure. Only 6 per cent of the children were observed before attendance at school in the first grade. The median age at admission to the clinic was 8 years to 8 years and 11 months, when average children would be in the third grade of school. The largest single age group, however

(24 per cent), already had manifest behavior difficulties at the age of 7 years to 7 years and 11 months.

The intelligence levels of the group are shown in table 2.

In a normal child population 60 per cent would have an intelligence quotient between 90 and 109, with 20 per cent having a quotient above and 20 per cent having a quotient below these normal levels. My figures

TABLE 2.—*Intelligence Levels*

I. Q.	Number	Per Cent
80- 89	6	12
90- 99	15	30
100-109	19	38
110-119	8	16
120 and over	2	4
Total	50	100

TABLE 3.—*Eye Dominance and Handedness*

Right Eye Dominant		Per Cent of Total Children
Right handed	0	0
Left handed	2	4
(Both partially converted, in that they wrote with the right hand)		
Right handed	12	24
(By conversion)		
Left handed	1	2
(By conversion)		
Total	15	30
Left Eye Dominance		
Left handed	9	18
(Five partially converted to right handedness in writing)		
Left handed	0	0
(By conversion)		
Right handed	3	6
Right handed	19	38
(By conversion)		
Total	31	62
Mixed Eye Dominance		
Right handed	4	8
(By conversion)		

indicate a normal percentage with a quotient above 110, a slight increase in the normal group (having a quotient from 90 to 109) and a decrease of those in the below average group, thus showing a slight skewing of the normal curve of distribution as a result of the mode of selection. It is clear, however, that whatever educational disabilities were found could not be attributed to insufficient intellectual machinery.

Table 3 classifies the children in terms of right or left eye dominance with relation to handedness. It shows that twice as many (62 per cent)

children had the left eye dominant as had the right eye dominant (30 per cent), with 8 per cent having uncertain or mixed eye dominance. It is striking that among the children with dyslexia there were none who were right eyed and right handed. Of the 31 with the left eye dominant, 19 (61 per cent) had become right handed by conversion from an original sinistrality. Of the 15 with the right eye dominant, 12 (80 per cent), similarly, were right handed by reason of conversion from left handedness. Of the 50 children, 18 per cent were left eyed and left handed but with partial conversion for writing, 6 per cent were left eyed and right handed, 4 per cent were right eyed and left handed with conversion for writing and 2 per cent were right eyed but left handed by conversion because of incapacity of the right hand. The significance of dominance of the left eye is demonstrated by the high proportion of children with the left eye dominant among the children with dyslexia.

It is noteworthy that the right-eyed children evidenced the result of crossed motor pathways, since unilateral dominance is lacking when dyslexia occurs, as shown by the 2 children right-eyed and left-handed but converted to right handedness for writing but not for other muscular effort. This effect is further emphasized by the 12 who were right handed from complete conversion from sinistrality, as well as by the 1 child who had been obliged to become left handed because of difficulty with his more effective right hand. These data suggest that the problem is not merely one of dominance of the right or of the left eye but originates in the total organization of the individual in terms of eye-hand association and coordination as related to the cortical organization of form material in its space relationships.

The symptoms presented by this group of children merit attention. All the children showed a primary confusion in dealing with symbol forms. This was manifest in the writing of figures, letters or words with reversals, in strephosymbolia, in mirror writing and in facile mirror reading. There was confusion in the perception, retention and reproduction of the forms of graphic language. Some of the children showed a marked disorientation with reference to right and left, and a few were uncertain of their postural status in movement. A considerable proportion had some limitation of speech, and a definitely high percentage exhibited marked infantilism. Fundamentally they all showed a lack of facility with forms, were weak in representation and indicated a poor power of association of visual and aural impressions.

These symptoms constituted the major ones primarily related to vision, but there were in addition the significant secondary behavior problems which arose from effects on the total personality and because of which the children were brought to the clinic for treatment. These included the behavior reactions of frustration, disappointment and inadequacy, accompanied more frequently by a compensatory aggression than by submission.

The growing personality is affected not merely because of the internal confusion in the learning processes but because the resultant educational defect is overt and, therefore, observable at home and exhibited in the classroom to the painful discomfort of the ego. The child suffers a severe emotional reaction to failure, but in addition he is humiliated by the deflation of the ego when he fails to acquire satisfactory social approbation through his effort at achievement in educational competition. This pattern accounts for the prevalence of tantrums and truancy, the restlessness and irritability, the lack of interest in reading, the disgust with school attendance and the discouragement at specific weakness in school subjects, with the accompanying retardation, nonpromotion, demotion or forced promotion to a higher class more revealing of the shortcoming. Among distressing forms of behavior are severe emotional reactions such as anorexia, crying, emesis, enuresis, disobedience, pugnacity, timidity, social withdrawal, unfriendliness and unhappiness. Antisocial reactions are shown in lying, stealing and running away from home. These constituted the prevailing behavior reactions for which the present group was being scolded, deprived, condemned and punished. The children's personality limitations and behavior problems were related mainly to the learning difficulties incidental to mental confusion arising from a lack of harmony in eye dominance and handedness.

The management of eye dominance may be divided into four main elements.

The first involves the reeducation of the family concerning the meaning of the child's behavior and its relation to eye-hand dominance. Regardless of the method of treatment employed, possibly 10 per cent of the children will make exceedingly poor progress because of the lack of family understanding and resultant poor cooperation or failure to facilitate regular and continued efforts at overcoming the educational problem. Family pride and ambition require careful readjustment, but so do the intrafamilial friction and emotionality.

The second line of treatment calls for the education of the school as to the nature and meaning of the scholastic inadequacy of the child, the setting up of a series of adjustments in the school program that will make provision for continued progress in terms of the intelligence quotient and the provision of remedial training, particularly in reading.

The third factor, and the first step, is the correction of all optical defects. It should be remembered that so far as is possible and wise, the better correction should be provided for the dominant eye, so that further confusion and strain will not be put on the child by sharper acuity of the nondominant eye. Supplying the proper correction per se does not cause any shift in eye dominance. It is almost needless to state that it is desirable to have the eye dominance of children determined prior to

admission to school. This course would forestall the possibility of visual confusion or at least make possible the early and most promising remedial efforts required to harmonize the dominant eye–hand relationship.

The fourth phase of treatment is concerned with the partial reorganization of the personality of the child. I shall not discuss the questions involved in the reconversion of a child to his original dominant handedness, as this would take me too far afield. In undertaking the personality readjustment it is obvious that one must be concerned with the functional problems growing out of eye dominance. Some general principles to be observed in therapy are applied in the following six procedures:

1. When the left eye is dominant and natural left handedness exists, the child should wear a patch over the left eye during part of the learning process at home. The wearing of a patch permits greater cerebral attention to the right eye forms involved in learning and promotes the right-eyed progression from left to right so essential for reading. It should be supplemented by tutoring through various remedial methods, at times including the kinesthetic approach to the learning of figure and letter forms.

2. When there is left eye dominance with natural right handedness, treatment consists principally in the use of a patch over the left eye supplemented by remedial teaching.

3. When the left eye is dominant and the right hand is generally employed for writing and other activities because of conversion from sinistrality, there may be an increased need for the kinesthetic approach to supplement the remedial reading through tutoring and the use of the patch over the left eye. Not infrequently it is necessary to return the child entirely to the original left-handed activity.

4. When the right eye is dominant and the right hand is employed by virtue of conversion from sinistrality, it is important to emphasize the kinesthetic approach. If there is any stammering it is usually desirable to reconvert the child to left handedness.

5. When the right eye is dominant and native left handedness is retained, a kinesthetic approach is often helpful, but remedial tutoring is essential.

6. The further management depends on making the child appreciate the nature of his disorder and eliciting his cooperation in carrying out the details of treatment. He requires support and encouragement as he progresses, through demonstration of his advancement in educational achievement. His advance is hastened by drills to promote the memory, retention, association and reproduction of forms. His increased power along these psychologic lines affords him direct evidence of his improved

organization for learning. This promotes greater interest and is a stimulus to greater effort.

These general methods were used in the management of the present group of 50 children, and only 4 (8 per cent) made poor progress. These 4 children were pugnacious and resentful, engaged in truancy and were beyond parental control, although 3 of them were between the ages of 7 years and 2 months and 7 years and 11 months. The others showed definite improvement, but it is striking that the most marked improvement was made by (*a*) the children whose right eye was dominant and who had been converted to left handedness because of an injury to the right hand and (*b*) those whose left eye was dominant and who were reconverted to their original left handedness.

The progress of many children is determined not wholly by whether the right or the left eye was dominant but also by the age at which treatment is undertaken, the level of the intelligence, the emotional stability, the regularity of remedial work, the effective employment of a patch over the left eye and, above all, continued intelligent tutorial work in reading, arithmetic and other school subjects. There is a direct gain through the correction of all remediable physical defects, the correction of lateral imbalance and the psychologic improvement of visual and aural mental representation through associational drills concerned with forms and symbols. Psychiatric guidance may be necessary for the child and the family.

Clearly the management of eye dominance should be a matter of interest to the ophthalmologist. He should make determination of the dominant eye a routine examination, even though he may have to advise tutorial assistance to supplement the glasses prescribed to secure a higher degree of visual acuity.

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NEUROPSYCHIATRIC GERIATRICS

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Philosophers devoted much of their time and energy to the difficult task of proving the obvious. That they succeeded well is evident, since even physicians are more familiar with Descartes' *Cogito, ergo sum* than they are with his pioneer experiments on optics and vision. Old age, too, is obvious, but its definition becomes elusive. It begins in youth and progresses at an uneven pace in various organs of the body. Certainly it is physiologic rather than chronologic, and its attendant loss of physical and mental resilience involves a process of breaking rather than of bending. One is never quite sure when a person has died of old age, but one knows when he has died of disease. As causes of illness and death in old age, diseases of thoracic and abdominal viscera and of blood vessels are strikingly frequent, but as causes of senile decadence, diseases of the nervous system, muscles and bones take first place. All of these and habitual indolence lead to impaired social usefulness; this is the criterion by which one segregates the young from the old. Psychiatric and democratic training makes one shun the facts of heredity, but they are inescapable and bear not only on the phenomenon of longevity but on the output of energy. The survival of any endogenous gifts is not assured, however, for they are subjected to the assaults of the environment, and the two should be separated carefully when one attempts to improve the fortunes of the aged.

The principal causes of illness and death in the rapidly increasing population of older persons are known fairly accurately. High in the list are cardiac disease, cancer, circulatory failure of the brain and accidents.[1]

SENESCENT MORBIDITY AMONG 200 PATIENTS

Before attempting to make a survey of the neuropsychiatric disorders encountered among other patients who were referred to the neurologic department for more detailed study, I sought to familiarize myself with the general background of senescent morbidity as seen at the Mayo Clinic. For this purpose the records of 200 patients of 60 years and

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1. Thewlis, M. W.: The Care of the Aged (Geriatrics), ed. 3, St. Louis, C. V. Mosby Company, 1941.

more, in the order of their registration and with due regard to the season of the year, were reviewed. There were 115 men and 85 women, which is the distribution seen in the clinic as a whole. The disorders noted most commonly are named in figure 1. Numerous other diseases, most of them not peculiar to old age, were spread more thinly among the older registrants.

The high incidence of neuroses was impressive and unexpected. Many of the patients had been seen over a period of years, and the same familiar complaints were noted in the older records. Well represented were the gastrointestinal neuroses, the depressions, chronic fatigue states and anxieties. One need hardly repeat that a diagnosis of neurosis

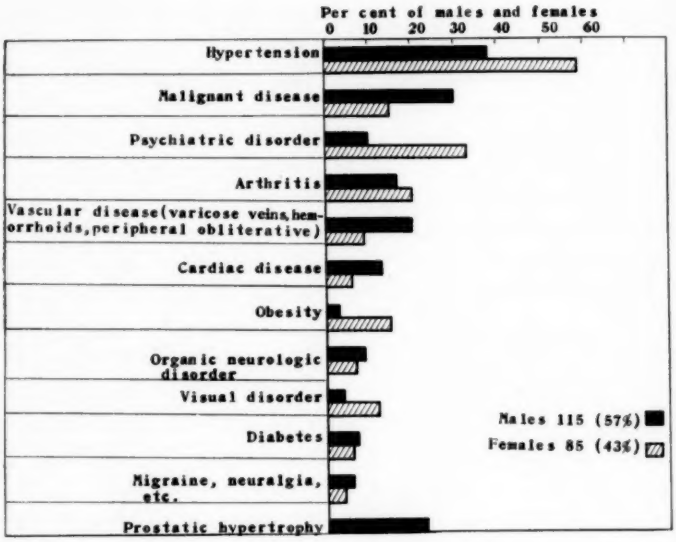

Fig. 1.—Most common disorders found among 200 patients aged 60 years or more.

should rest on good evidence. Alvarez [2] expressed this admonition by saying, "When faced by the elderly man or woman who has suddenly become ill, we physicians must be conscientious pessimists and not careless optimists."

Impairment in hearing is recognized as a common affliction of the old. Complaints of headache and vertigo or giddiness that could be related to the aging process were not prominent and in most instances had been mentioned in antecedent records. It has been said facetiously that migraine is a type of recurrent headache that is supposed to dis-

2. Alvarez, W. C.: The Care of the Aged, News-Letter, Englewood Branch, Chicago M. Soc., January 1938, vol. 30.

appear as a patient grows older, but it almost never does. Of 9 patients concerning whom a diagnosis of migraine had been made, and doubtless many more had had it, 6 had ceased to complain of it; more of the 6 were women than men.

PATHOLOGIC CHANGES IN SENESCENCE

Vascular accidents are an important cause of disability among the aged, but they do not represent primary neural involutional phenomena. Since the assignment of neurologic disabilities to either of these specific pathologic changes is extraordinarily difficult, it is helpful, as Critchley [3] did in his excellent and informative Goulstonian Lectures on "The Neurology of Old Age," to review the pathologic changes before discussing the clinical aspects.

Many of the changes that take place in old age have been known for years. The skull is often thickened, especially in the frontal region, but it may be thinned. Whether these changes are primary or secondary to alterations in the nervous system itself is not known. The dura may be thickened and become the seat of calcareous deposits. The leptomeninges also may be thickened, but usually they can be stripped off the hemispheres readily. The brain may be shrunken and feel unduly hard; the sulci, deepened and widened, especially in the frontal lobes. The ventricles may be dilated, their walls studded and ridged, and the lost tissue replaced by an increased quantity of cerebrospinal fluid. The cortical gray markings may be less distinct and thinner than usual; the white matter and the deep nuclei, shrunken in size.

Arteriosclerotic changes, when present, are most obvious in the larger vessels at the base, and areas of infarction, both large and small, may be found. Changes incidental to hypertension are usually widespread and include foci of hemorrhage, edema, gliosis and cellular infiltration.[4]

The bodies of the nerve cells may be atrophied, laden with pigment, vacuolated and decreased in number, and their processes also may be reduced in number and replaced by gliosis. Corpora amylacea are common and especially abundant under the ependyma. Senile plaques may be present. Much has been written on their origin and meaning.[5] Critchley summarized the present conception regarding them as follows:

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3. Critchley, M.: The Neurology of Old Age (Goulstonian Lecture), Lancet **1**:1119-1127 (May 23); 1221-1230 (June 6); 1331-1336 (June 20) 1931.

4. Rosenberg, E. F.: The Brain in Malignant Hypertension: Clinicopathologic Study, Arch. Int. Med. **65**:545-586 (March) 1940.

5. Soniat, T. L. L.: Histogenesis of Senile Plaques, Arch. Neurol. & Psychiat. **46**:101-114 (July) 1941.

Senile plaques occur in the normal senium provided the individual had attained a very great age (? over 90); they occur very sparsely, however, so long as the mental condition is good. In cases of senile psychosis they appear at a much earlier age and may be very numerous indeed; cases of dementia in old age occasionally show no plaques, and such cases may well belong to a psychological category of their own. In Alzheimer's psychosis plaques occur in great numbers and at a relatively youthful age.

Changes in the choroid plexus are commonly noted, but how, and whether, they affect the function of the brain is unknown.

In the spinal cord similar changes take place, although here senile plaques do not occur. An alteration that may be noteworthy is marginal pallor of the cord, especially of the fasciculus gracilis, as it appears in the sections stained by Weigert's technic. What this may mean from a functional standpoint is not clear.

The peripheral nerves, from their roots outward, may suffer a reduction in the number of fibers and an increase in the supportive tissues, changes that need more study since they probably are related to some of the complaints made by old persons and to some of the sensory changes noted in old age.[6]

The reader will get the impression that these changes are widespread. This is correct. He would be likely to draw the inference that they explain in a satisfactory manner the neuropsychiatric picture of old age. Unfortunately there is no clear correlation between the intensity of the clinical disability and the pathologic changes.

NEUROLOGIC DISTURBANCES AMONG 200 SENESCENT PATIENTS

The principal neurologic disturbances noted among patients who were referred to the neurologic department for more detailed study are recorded in figure 2.

Peripheral Nerve Involvement.—Fifty-six patients (28 per cent) gave evidence of involvement of the peripheral nerves, the sciatic nerve being the one most commonly affected. There were 19 cases of sciatic neuritis; most of these cases were of the type that usually is encountered among younger persons. Herpes, which was present in 8 cases, is particularly distressing, since the pain commonly persists for years. The injection of autogenous blood, reserved for cases of recent involvement, roentgen therapy and the administration of cobra venom are often unavailing. Excision in the area and skin grafting have been used successfully in 2 instances by Peyton.[7] In 6 cases the picture was

6. Corbin, K. B., and Gardner, E. D.: Decrease in Number of Myelinated Fibers in Human Spinal Roots with Age, Anat. Rec. **68**:63-74 (April) 1937. Cottrell, L.: Histologic Variations with Age in Apparently Normal Peripheral Nerve Trunks, Arch. Neurol. & Psychiat. **43**:1138-1150 (June) 1940.

7. Peyton, W. T.: Unpublished data.

that of polyneuritis; in 2 of these the condition was classified as senile; it appeared to be due in 1 to cold, in 1 to alcohol and in 1 to a deficiency, and in 1 the cause remained unknown. In 6 other cases the neuritis was classified as diabetic in origin. Of the 4 instances of cranial nerve palsy, in 3 the seventh nerve was involved; in 2 of these the condition was due to a malignant process of the parotid gland. Brachial neuritis

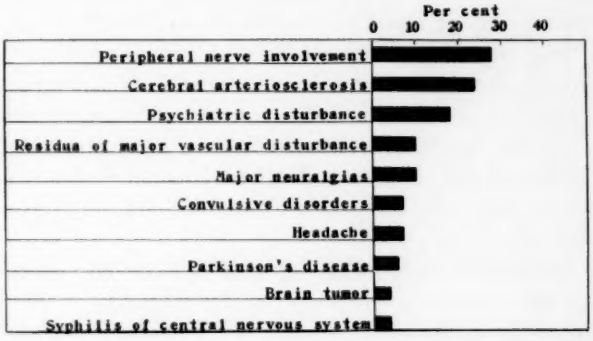

Fig. 2.—Principal neurologic disturbances noted among 200 patients aged 60 years or more who were referred to the neurologic department.

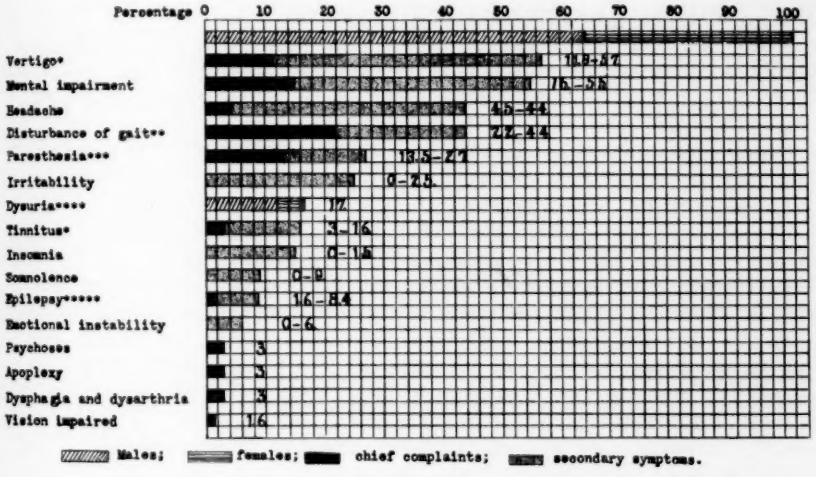

Fig. 3.—Disabilities of 59 patients concerning whom a diagnosis of cerebrospinal arteriosclerosis had been made. * Patients with the mildest type excluded. ** Weakness, ataxia, spasticity, shuffling, spasticity with ataxia, and tremor, respectively. *** Numbness 10 per cent (hands and feet); burning 6 per cent (feet, back, hips); coldness 3 per cent (feet and knees); glossodynia 1.5 per cent. **** Proportion of men to women as 8 is to 3; of men alone, dysuria was the chief complaint in 21 per cent. ***** Two of the 5 had petit mal attacks.

occurred in 4 cases, twice as a complication of surgical operation and once as a result of carcinomatous invasion of the plexus. In 3 instances there was radiculitis. While peroneal nerve palsy was noted only twice, the "crossed knee" variety occurs most commonly among elderly undernourished men.

Cerebral Arteriosclerosis.—The diagnosis of cerebral arteriosclerosis usually is made in cases in which there is a history of vascular episodes. This important group of cases warranted a special survey. It has been emphasized long and repeatedly that the findings noted on palpation of the peripheral vessels do not help greatly in establishing a diagnosis of cerebral arteriosclerosis or atherosclerosis. The exact allocation of symptoms either to cerebral arteriosclerosis or to diffuse senile parenchymatous degeneration is often difficult and is untrustworthy at best. The differential diagnosis from other diseases, such as tumor of the brain, may be impossible.

With the thought in mind that involvement of the spinal cord as well as of the brain would be helpful in lending support to the diagnosis of what usually passes as arteriosclerosis of the nervous system, I studied the records of 59 additional patients with "cerebrospinal arteriosclerosis." Sixty-four per cent of them were men and 46 per cent women. The extremes of age were 50 and 78, the average age being 63. The blood pressure of 64 per cent of the patients was normal. The disabilities (fig. 3) and findings (fig. 4) in such a selected group are obviously more severe and more striking, respectively, than those in patients for whom the diagnosis is simply cerebral arteriosclerosis.

An exemplary case of the disorder was that of a man aged 52 who complained principally of incontinence, which had been growing worse for two years. Coincident with this was a general failure of strength and the development of a tremor in the lower extremities. One year before his examination, he had begun to have dull, heavy headaches and a sensation of having his hat on. His friends noticed that his memory was failing. He left the harnessed horses to stand in the field all night and forgot to take off their bridles when they were to eat; he expected to dine shortly after a meal was over and became badly confused in giving the ages of his children. He became dizzy easily, often fell asleep during the day, was confused in a crowd and often became lost in his own house after he had been lying down. At night he interpreted the rumbling of horses over a nearby bridge as thunder. The neurologic findings included evidence of combined sclerosis of the spinal cord.

Among these 59 patients the most commonly occurring chief complaint was a disturbance of gait. "Senile paraplegia" is a descriptive term covering a variety of lesions. Critchley classified these as cortical, subcortical and spinal. The first is primarily a pyramidal and prepyramidal disorder. Scattered lesions throughout the deeper structures of the brain, often arteriopathic in origin, give rise to the subcortical variety

and may produce pseudobulbar and parkinsonian disabilities. Both cortical and subcortical types may be accompanied by a variety of incontinence in which the discharge of urine or feces, once it has started, is beyond control. Such incontinence also may be associated with ataxia of cerebellar origin. In some cases the incontinence is related to a coexisting mental deterioration. The spinal variety is essentially myelic, and the findings are usually those of combined sclerosis. In a few instances the onset of the disability is apoplectiform. It should not be overlooked that extraneural myosclerotic processes also impair the gait and that such muscular changes may also become the basis of sphincteric incompetence. There are also striking disturbances in gait that are not easily explained on the basis of involvement of any particular structure of the nervous system, such as walking with tiny steps, the marche à petits pas, and a singularly distinctive hesitation in walking which can be overcome by sufficient coaxing, Petrén's gait.

In this group of 59 patients a commonly occurring complaint was of giddiness rather than vertigo. Intense vertigo, like that seen most commonly in association with Ménière's disease, occurs less often and suggests a lesion of the brain stem, such as thrombosis of the posterior inferior cerebellar artery. As a cause of giddiness among older persons, increased sensitivity of the carotid sinus also may come into play.

Paresthesia, especially numbness, burning and coldness of the extremities, occurred commonly and in some instances may well have been related to impairment of circulation. Patients who have a persistent, symmetric burning of the tongue and inside of the mouth, unaccompanied by any objective findings, to which has been given the name "glossodynia," usually are women beyond the age of 50 and often have associated neuroses and depressions.[8]

Headache, when present, was usually bregmatic or frontal in situation and generally of a dull, heavy type, but sometimes stabbing and sharply localized and not greatly influenced by straining or jarring of the head. An unfruitful attempt was made to learn whether so-called arteriosclerotic headache might have some pathognomonic characteristics. To this end a review was made of the records of 100 patients who had come to necropsy and concerning whom a diagnosis of cerebral arteriosclerosis had been made by the pathologist. Two thirds of these patients had had hypertension, and the remainder had not. However, of each group a third had had headache.

The neurologic findings among the 59 patients who are the immediate concern here were fairly characteristic. The pupils were often somewhat irregular and sluggish in their reactions, and conjugate rotation of the globes upward might be limited. While testing sensation a striking observation was poorly sustained attention. Thus, when

8. Gilpin, S. F.: Glossodynia, J. A. M. A. **106**:1722-1724 (May 16) 1936.

asked to respond by saying "sharp" or "dull" each time the point or the head of a pin was applied, or to announce whether or not they could feel the vibrations imparted by a tuning fork, many patients presently failed to answer at all or dropped into the habit of repeating the same response rhythmically. The observations were usually more reliable when appreciation of temperature was tested. Appreciation of vibratory sensibility was commonly impaired. At what level of the nervous system the various sensory impulses are arrested is not clear.

Psychiatric Disturbances.—Socially and economically the greatest problem related to old age is psychiatric. Dayton, whom Overholser [9] quoted, brought out the fact that mental disorder, far from being a disease of youth, is primarily one of old age.[10] Many of the patients are withdrawn from the general population because of conduct intolerable from a social point of view: indecent exposure, a persistent tendency to wander away while confused, a tendency to set fires or to cause serious danger to themselves or to others and untidiness, restlessness and noisiness, which create trying nursing problems.[10] It is not unlikely that most of such mental disorders are primarily constitutional and only secondarily due to degenerative diseases, such as cerebral arteriosclerosis. Superimposed processes must occur often in old age. That they do is suggested also by the findings noted in examination of the spinal fluid. In the presence of senile and arteriosclerotic psychoses, Bruetsch [11] often found elevation in the concentration of protein in the spinal fluid, especially among men. In some cases this observation was followed by disclosure at necropsy of regions of recent destruction in the brain.

Among the psychiatric disorders noted in the group of patients under consideration here (fig. 2), the neuroses, as mentioned before, were prominently represented. These were followed in frequency by the depressive and the delirious and confusional states and these, in turn, by psychoses more often thought of as belonging to the senile group.

Simple senility, or dotage, is by no means inevitable to all old persons, but it is generally regarded as being within normal limits. There is a tendency to forgetfulness, to living in the past and to loss of fluidity of interests. Often there is a hypochondriacal concern over bodily functions. It is related that Voltaire and an old crony entered

9. Overholser, W.: Problems of Mental Disease in an Aging Population, in Proceedings of the National Conference on Social Work, New York, Columbia University Press, 1941, pp. 455-463.

10. Overholser, W.: Some Mental Problems of Aging and Their Management, M. Ann. District of Columbia **10**:212-217 (June) 1941.

11. Bruetsch, W. L.; Bahr, M. A.; Dieter, W. J., and Skobba, J. S.: The Cerebrospinal Fluid Total Protein in the Psychoses: The Results of Spinal Fluid Examinations in 2,823 Mental Patients (Exclusive of the Syphilitic Psychoses), Dis. Nerv. System **2**:319-324 (Oct.) 1941.

into a pact to commit suicide, but the event was called off on the following morning when one of them had had a good bowel movement. The senile psychoses are fairly well defined and are usually of gradual onset, marked by forgetfulness, especially for recent experiences; defects in orientation; aimless hyperactivity; reduction in attention, concentration and thinking; often a self centering of interest; stubborn opposition; fabrication, and paranoid trends. Pick's disease is a chronic form of dementia which is progressive, accompanied by focal manifestations and characterized pathologically by gross, circumscribed, symmetric regions of atrophy, especially of the frontal and temporal lobes of the brain. Alzheimer's disease resembles senile dementia but occurs earlier and is more rapid in its progress. There is a more generalized atrophy of the brain, which is accompanied by severe and characteristic histologic changes in the cortex. The causes of these psychoses remain uncertain.

Vascular Disturbances.—The abrupt onset of vascular accidents to the brain assists materially in their recognition. Not always recognized, however, is the relationship of dull, disturbing, persistent unilateral pain in the limbs or the abdomen, which may appear gradually after a stroke that involves the sensory pathways. There is no satisfactory method of relieving the discomfort of this "thalamic syndrome," but its intensity fluctuates greatly with the mood of the patient, which gives the most satisfactory clue to treatment. As drivers of automobiles, patients having a recently acquired hemianopsic defect are probably more dangerous to the public and to themselves than those whose vision is impaired through other causes.

Parkinsonism.—Perhaps the commonest clinical features of old age are the extrapyramidal symptoms. The changes to which I refer are in part postural. To the attitude of flexion associated with rigidity are added components of muscular and skeletal origin, which all together contribute to slowness and poverty of movement. The extrapyramidal system is notoriously prone to partnership in heredofamilial diseases and in what Gowers so aptly called "abiotrophy," that is, a limited capacity for survival. Thus, a part only of the nervous system may be singled out for involutional changes. The identification of Parkinson's disease, encephalitic parkinsonism and arteriosclerotic parkinsonism or features solely related to the parkinsonian syndrome and exclusive of the more obvious characteristics of encephalitis and the arteriopathies, I do not find easy.

The so-called senile or arteriosclerotic tremor of the head, hands, arms and other parts of the body is often "essential" or heredofamilial. The history may then indicate that the tremor has been present for years, especially under conditions of fatigue, and that other members of the family have been affected.

Tumor of the Brain.—Among older patients the diagnosis of tumor of the brain presents more than the usual difficulties. Mental symptoms often classified as "organic dementia" are of comparatively common occurrence; headache and choked disks, comparatively uncommon. In a series of 100 verified cases of tumor of the brain among patients 60 years of age or more,[12] mental symptoms were given as the chief complaint in 18 per cent and headache in 15 per cent, and choked disks were found in 33 per cent. Glioblastoma multiforme, meningioma and acoustic neuroma, in the order named, made up 82 per cent of the tumors in the series. Medulloblastoma and astrocytoma, common among children, are not found or are rarely found among the aged.[13] Glio-

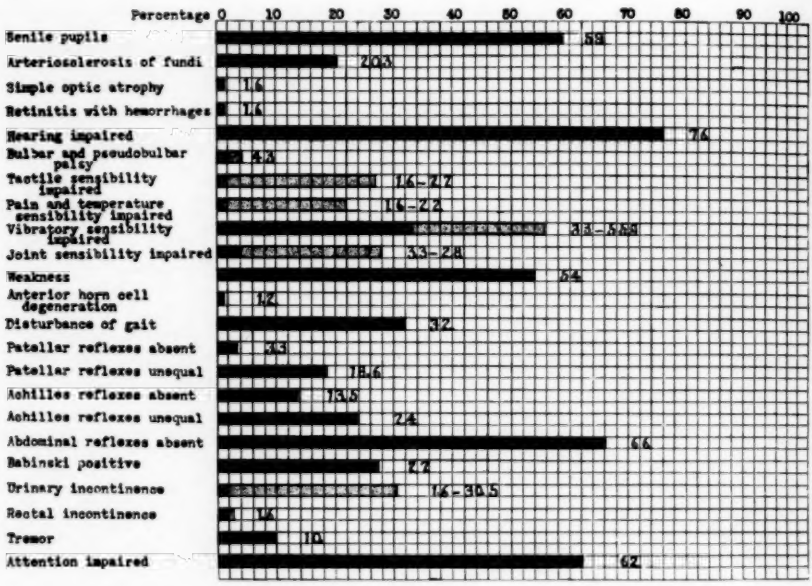

Fig. 4.—Signs of disease among 59 patients concerning whom a diagnosis of cerebrospinal arteriosclerosis had been made. Solid bars, chief complaints; dotted bars, secondary symptoms.

blastoma multiforme, rare among children, is common among the aged, and the operative mortality among the aged, especially when there is no choking of the disks, is high. Choking of the disks, also, becomes less frequent with advancing years in association with chronic intradural hematomas.

12. Moersch, F. P.; Craig, W. M., and Kernohan, J. W.: Tumors of the Brain in Aged Persons, Arch. Neurol. & Psychiat. **45**:235-245 (Feb.) 1941.

13. Bailey, P.; Buchanan, D. N., and Bucy, P. C.: Intracranial Tumors of Infancy and Childhood, Chicago, University of Chicago Press, 1939.

THE NEED FOR MORE KNOWLEDGE

There is need for a better understanding of the manner in which the nervous system functions and for greater knowledge bearing on the causes, prevention and cure of the neuropsychiatric disorders of the aged. Such understanding and knowledge are not to be accomplished by the introduction of a "tyranny of words." Proper emphasis already has been placed on the supportive functions of circulation and nutrition. The neural apparatus itself must be sound and possess lasting qualities. This is looking for a great deal in "the most complicated instrument known to science." All one can do at present is to take good care of it. One must practice temperance. One can and must foster the freedoms in this country by preventing the frustration of the aged caused by enforced early retirement, dependency and isolation. Experience begets judgment and wisdom, and of these there has never been a surplus. These are contributions older persons can make. Expansion of the family care system, residence in domiciliary institutions and provisions for some degree of financial independence through the granting of pensions will restore self respect and provide prospects that are not too sear and forbidding. All assets are seldom lost through age alone. Too often they are terminated by illness and neglect.

The Mayo Clinic.

SIMPLE POSTERIOR SCLEROTOMY AND SCLERECTOMY

AN ADJUNCT TO OPERATIONS FOR GLAUCOMA

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NEW YORK

Incision of the posterior part of the sclera is one of the oldest of operative measures for the relief of increased intraocular pressure. According to Priestley Smith,[1] Beard [2] and Duke-Elder [3] it was William MacKenzie of Glasgow who first made use of this procedure, around 1830. However, Masselon,[4] writing in 1886, stated that Guerin, of Lyon, had advised scleral puncture for ocular hypertension more than one hundred years previously. After MacKenzie this procedure seems to have fallen into disuse until revived by de Luca.[5] Thereafter it was recommended in turn by such outstanding ophthalmic surgeons as Weber,[6] Nicati,[7] Parinaud,[8] Priestley Smith [1] and Gifford.[9]

Beard [2] also stated that it was Masselon [4] who first conceived the idea of doing a simple posterior sclerotomy without choroidotomy or retinotomy. But a rereading of the latter's concise French seems to show that he was more interested in sparing the vitreous than in avoiding injury to the choroid and the retina. For in describing his "sclérotomie équatoriale" he advised that a flat knife be passed between the external and the inferior rectus muscle behind the ciliary zone and "sous les enveloppes de l'oeil . . . en rasant autant que possible la surface de l'humeur vitrée."

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1. Smith, P.: On Puncture of the Sclera as an Adjunct to Iridectomy in the Treatment of Glaucoma, Tr. Eighth Internat. Ophth. Cong., Edinburgh, 1894, p. 33.
2. Beard, C. H.: Ophthalmic Surgery, ed. 2, Philadelphia, P. Blakiston's Son & Co., 1914, p. 420.
3. Duke-Elder, W. S.: Text-Book of Ophthalmology, St. Louis, C. V. Mosby Company, 1941, vol. 3, p. 3401.
4. Masselon, J.: Sur le glaucome, Ann. d'ocul. **95**:231, 1886.
5. de Luca, cited by Beard.[2]
6. Weber, A.: Die Ursache des Glaukoms, Arch. f. Ophth. **23**:1, 1877.
7. Nicati, W.: Note sur la guérison du glaucome par la sclérotomie équatoriale transverse, Bull. et mém. Soc. chir. de Paris **7**:622, 1881.
8. Parinaud, H.: Traitement du glaucome par la paracentèse sclérotique, Arch. d'opht. **5**:180, 1885.
9. Gifford, H.: Posterior Sclerotomy Preliminary to Glaucoma Operations, Ophth. Rev. **12**:248, 1893.

Therefore it is Simi [10] or, more probably, Galezowski [11] to whom credit for the first simple posterior sclerotomy is due. The latter first described his "sclérotomie postérieure" for glaucoma at the seventh International Ophthalmologic Congress at Heidelberg in 1888. His method was to pass a flat knife between the sclera and the choroid for several millimeters meridionally, starting well behind the ciliary body. He made two such incisions, one between the superior and the external rectus muscle and another between the inferior and the external rectus muscle. In describing his procedure he added rather optimistically that "la maladie se trouve complétement arretée par cette operation."

Simple posterior sclerectomy, i. e. excision of a segment of sclera, was first done by Argyll Robertson,[12] who in 1876 reported 4 such operations. He removed a circular piece of sclera "two lines from the limbus" with a $\frac{1}{12}$ inch (0.2 cm.) Bowman trephine, thus "permitting the escape of some of the superabundant fluid in the chamber of the vitreous humour." Despite his belief that "the reduction in tension thus produced is likely to be of a permanent character," he had few followers. Only Fröhlich,[13] in 1904, reported permanent relief of hypertension, inflammation and pain in 4 of 5 cases of absolute glaucoma following posterior trephining of the sclera. He used a 5 mm. von Hippel trephine to remove a segment of sclera from behind the ciliary body and between the external and the inferior rectus muscle.

COMMENT

Since the introduction of these early procedures, long and bitter experience has shown that no operation on the posterior scleral segment of the eye will alone reduce intraocular pressure permanently. Whatever success has been attained in the surgical treatment of glaucoma has been due to procedures aimed at increasing the filtration of aqueous, i. e., operations on the anterior segment. Priestley Smith [14] and Gifford [9] recognized this fact long ago when they suggested puncture of the sclera as an adjunct to iridectomy rather than as a permanent operation per se, as recommended by Robertson,[12] Masselon,[4] Galezowski [11] and others. In recent years it has been the usual custom to do anterior

10. Simi, A.: Puntura della sclerotica nel glaucoma, contributo allo studio della oftalmotomia posteriore, Boll. d'ocul. **9**:17, 1887.

11. Galezowski, X., in discussion on Wohlfors, K. R.: Ueber Druck und Druckmessungen in menschlichen Auge, Tr. Seventh Internat. Ophth. Cong., Heidelberg, 1888, p. 282.

12. Robertson, D. A.: Trephining the Sclerotic: A New Operation for Glaucoma, Ophth. Hosp. Rep. **8**:404, 1874-1876.

13. Fröhlich, K.: Ueber die Trepanation der Sklera bei Schmerzhafter Glaukomblindheit, Klin. Monatsbl. f. Augenh. **42**:411, 1904.

14. Smith, P.: On the Pathology and Treatment of Glaucoma, London, J. & A. Churchill, 1891; footnote 1.

(aqueous chamber) or—more commonly—posterior (vitreous chamber) paracentesis in order to decrease the tension sufficiently to allow a more permanent surgical procedure to be done with greater safety.

It is axiomatic that an operation for glaucoma is safest when done on an eye in which the tension is as near normal as possible. And it is to attain such a condition that the posterior through and through sclerotomy has had its main use in cases in which reduction of tension has not been the response to use of miotics and to intravenous or retro-orbital injections. As every ophthalmologist knows, some of the more important reasons for sclerotomy are to reduce the risk of expulsive hemorrhage, to encourage formation of a deeper anterior chamber, to relieve excruciating pain when enucleation is not possible or desirable and to allow time for preparation for a more permanent operation. These are all good and valid reasons.

However, there are also some possible objections to this procedure. The most important is that when there is some hope of ultimate retention of useful vision, especially in the case of recalcitrant acute primary glaucoma, paracentesis of either the aqueous or the vitreous chamber, it has always seemed to me, affords at least as great a possibility of sudden release of pressure with a consequent expulsive hemorrhage as does a basal iridectomy or a trephining. Furthermore, the posterior sclerotomy which is most commonly used always carries with it the certainty of destruction of choroid and retina as well as the possibility of hemorrhage into the vitreous. No matter how unimportant these possibilities are theoretically they might better be avoided if another method can be found. Another objection is that both corneal puncture and scleral puncture often leave the eye so soft as to preclude any other immediate procedure, should this be desired, and necessitate a second operation later.

These facts were borne in mind when the following work was undertaken, the object being a reevaluation of some of the earlier fundamental procedures which might be of use as adjuncts to more permanent operations for glaucoma and would be less radical in their reduction of high intraocular pressure. In common with other glaucoma clinics, that at Bellevue Hospital receives some patients with acute and some with absolute glaucoma of primary and of secondary origin for which all conservative measures are useless. Such measures seem especially to be useless in the treatment of secondary glaucoma with total posterior synechias which has been long neglected and in which ordinarily the eye finally comes to enucleation. The absolute glaucoma of aphakia is also notoriously resistant to conservative treatment. Most of the work reported was done with such types. The procedures detailed hereafter

are called "simple posterior sclerotomy" and "simple posterior sclerectomy" as distinct from the penetrating posterior sclerotomy which includes both a choroidotomy and a retinotomy.

TECHNIC

After suitable infiltration anesthesia had been induced, the conjunctiva was incised from 3 to 9 o'clock 10 mm. above and parallel with the upper limbus. Together with the subconjunctival tissue it was undermined sufficiently so that the anterior flap could be reflected forward easily. A suture was passed beneath the superior rectus muscle so that the eye could be rotated downward. The sclera to each side of the muscle was then exposed by blunt dissection for about 10 mm. posterior to the insertion of the muscle. At the first operation a type A sclerotomy (fig. 1*A*) was done on both sides of the superior rectus muscle. This consisted

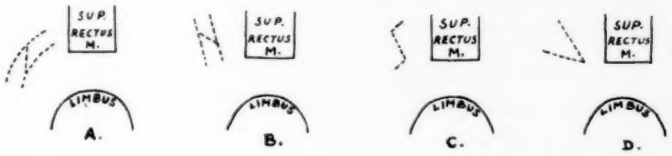

Fig. 1.—Types of simple posterior sclerotomy.

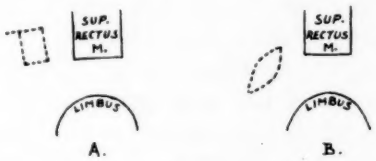

Fig. 2.—Types of simple posterior sclerectomy

of two parallel scleral incisions 8 mm. long and 1.5 mm. apart lying 10 mm. above and concentric with the limbus. Each was situated 2 mm. lateral to the muscle. These were then connected by a diagonal incision as indicated. The choroid immediately herniated through and the tension dropped (table 1). Three other types of sclerotomy were subsequently tried after similar preparation (fig. 1).

At the second operation, a type A sclerectomy (fig. 2*A*) was done on both sides of the superior rectus muscle. It consisted of the excision of two rectangles of sclera 2 by 5 mm. As indicated in figure 2*A*, these were subsequently enlarged by extending the posterior equatorial incisions to obtain a further drop in tension (case 2). Later the oval type of sclerectomy (fig. 2*B*) was adopted.

At first a discission knife was used to cut the sclera. However, it was soon found that all procedures could be done more safely and quickly by using fine sharp scissors as follows:

After the area to be incised or excised is outlined, the sclera is partially penetrated at one point with a sharp knife for a few millimeters and a suture is

inserted through one of the cut edges. In this way the globe is more easily controlled by using this suture in countertraction to the suture previously placed under the superior rectus muscle and held by an assistant to pull the eye downward. The scleral incision is then carried through to the choroid with the knife for a distance of 2 or 3 mm. This permits the insertion of a scissors blade, with which the operation is completed. Traction on the suture helps to raise the sclera slightly and prevents injury to the subjacent choroid. In the first four operations vitreous was lost twice. But after the adoption of scissors to complete the incision, vitreous was lost only once in thirteen operations.

As shown in figures 1 and 2, various types of sclerotomies and sclerectomies were tried, but type D sclerotomy and type B sclerectomy were finally adopted, since they were the simplest to do and produced as much lowering of tension as the more complicated procedures. All incisions were so placed that the anterior limits were well behind the ciliary body. When trephining or a modified Lagrange operation was to be done at the same time, the conjunctiva was undermined forward to the limbus, as is usually done.

After the first few operations several facts became apparent. It was found that a simple small incision or a small (2 mm.) trephining in the posterior portion of the sclera caused no appreciable reduction in tension. The scleral incision or excision must be large enough to permit choroid to herniate through freely. It was also learned that a single sclerotomy or sclerectomy of good size was more effective than two smaller excisions or incisions. Thus in case 2 the sclerectomies were too small and had to be enlarged by further scleral incision to cause sufficient drop in tension. Subsequent sclerectomies were made to measure 8 to 10 mm. in length. The type B oval sclerectomy usually measured 10 mm. in the long diameter and 4 to 5 mm. across the widest point.

The type of incision—whether radial or equatorial, straight or curved—was found to be unimportant providing the incision was large enough. On the other hand, there did seem to be a limit to the amount of drop in tension which could be obtained. Thus in operation 6 (case 4) the tension dropped from 56 to 19 after the temporal sclerectomy. No further drop occurred after the nasal scleral excision was made. In fact, except when vitreous was lost, the tension never dropped below the so-called normal level. Hence in all the following cases only one sclerotomy or sclerectomy was done. It was placed in the upper temporal quadrant between the superior and the external rectus muscle, this being the most easily accessible area.

A helpful feature of these procedures was that the tension could be taken at any time and the incision enlarged slightly to attain a further diminution if desired. The following case histories are epitomized in tables 1 and 2.

TABLE 1.—*Effect of Simple Posterior Sclerotomy and Sclerectomy on Tension* *

Operation No.	Diagnosis	Procedure	Preoperative Tension	Postoperative Tension	Drop in Tension
1	Absolute glaucoma	Bilateral† type A sclerotomy	80	35	45
2	Absolute glaucoma	Bilateral† type A sclerectomy	90+	48	42+
3	Absolute glaucoma	Bilateral† type B sclerotomy	90	Eye soft; vitreous lost	
4	Absolute glaucoma	Bilateral† type B sclerotomy	56	29	27
5	Absolute glaucoma	Type C sclerotomy	65	Eye soft; vitreous lost	
6	Absolute glaucoma; aphakia	Bilateral† type B sclerotomy	56	19	37
7	Acute glaucoma	Type B sclerectomy	90	35	55
8	Acute episode of chronic glaucoma	Type B sclerectomy	65	22	43
9	Secondary glaucoma; old uveitis	Type B sclerectomy	65	35	30
10	Absolute glaucoma; aphakia	Type B sclerectomy	65	40	25
11	Absolute glaucoma; secondary cataract	Type B sclerectomy	90+	Eye soft; vitreous lost	
12	Absolute glaucoma; old uveitis	Type D sclerotomy	90+	54	36+
13	Absolute glaucoma	Type B sclerectomy	76	35	41
14	Absolute glaucoma	Type D sclerotomy	68	29	39
15	Secondary glaucoma; senile cataract	Type B sclerectomy	65	30	35
16	Absolute glaucoma; aphakia	Type D sclerotomy	60	35	25
17	Chronic congestive glaucoma	Type D sclerotomy	65	40	25

* Expressed in millimeters of mercury as measured wtih the Schiøtz tonometer.
† On each side of the superior rectus muscle.

TABLE 2.—*Course and Results After Combined Procedures*

Case No.	Diagnosis	Procedures	Complications	Time Followed	Course and Results
1	Absolute glaucoma	Bilateral* type A sclerotomy	None	18 days	Pain and tension controlled
2	Absolute glaucoma	Bilateral* type A sclerectomy	None	4 wk.	Pain and tension not controlled
		Type C sclerotomy	Vitreous lost	4 wk.	Pain relieved but tension only partially controlled
3	Absolute glaucoma	Bilateral* type B sclerotomy	Vitreous lost	3 wk.	Pain and tension not controlled
		Bilateral* type B sclerotomy	None	4 wk.	Pain relieved; tension not controlled
4	Absolute glaucoma; aphakia	Bilateral* type B sclerotomy	None	3 wk.	Pain relieved; tension uncontrolled
5	Acute glaucoma	Type B sclerectomy; basal iridectomy	None	1 yr.	Pain and tension controlled
6	Acute episode in chronic glaucoma	Type B sclerectomy; trephining	None	11 mo.	Pain and tension controlled
7	Secondary glaucoma; old uveitis	Type B sclerectomy; modified Lagrange operation	None	10 days	Pain and tension controlled
8	Absolute glaucoma; aphakia	Type B sclerectomy; cyclodialysis	None	4 mo.	Pain and tension controlled
9	Absolute glaucoma; secondary cataract	Type B sclerectomy	Vitreous lost	5 wk.	Pain relieved; tension only partly controlled
10	Absolute glaucoma; old uveitis	Type D sclerotomy; basal iridectomy	None	6 mo.	Pain and tension relieved; miotics used
11	Absolute glaucoma	Type B sclerectomy; basal iridectomy	None	9 mo.	Pain relieved; tension uncontrolled
12	Absolute glaucoma	Type D sclerectomy; modified Lagrange operation	None	8 mo.	Pain relieved; tension controlled with miotics
13	Secondary glaucoma; senile cataract	Type B sclerectomy; basal iridectomy	None	6 mo.	Pain and tension controlled
14	Absolute glaucoma; aphakia	Type D sclerotomy; cyclodialysis	None	9 days	Pain and tension controlled
15	Chronic congestive glaucoma	Type D sclerotomy; modified Lagrange operation	None	6 wk.	Pain and tension controlled

* On each side of the superior rectus muscle.

REPORT OF CASES

CASE 1.—N. P., a white man aged 45, was referred by the medical service on Sept. 27, 1940 for pain in the right eye. Examination disclosed absolute glaucoma with a tension of 90 in the right eye and chronic simple glaucoma with a tension of 29 in the left eye. The right eye did not respond to use of miotics or to retrobulbar injection, and on October 3 a bilateral (on both sides of the superior rectus muscle) type A sclerotomy was done in the right eye, and the tension dropped from 80 to 35. The postoperative course was uneventful, and the patient was discharged back to the medical service on October 21 with the tension in the right eye 15 and that in the left still 29. He left the hospital shortly thereafter and did not return to the clinic for follow-up observation.

CASE 2.—J. K., a Negro man aged 41, was referred by the medical service for pain in the left eye on Oct. 15, 1940. A diagnosis of absolute glaucoma was made. The tension was 17 in the right eye and 90 + in the left. The eye did not respond to conservative measures, and on October 15 a type A sclerectomy was done on each side of the left superior rectus muscle, two pieces of sclera 2 by 5 mm. being excised. The tension dropped only to 70, and the scleral incisions were extended as shown in figure 2 *A*. The choroid herniated through more strongly, and the tension dropped to 48. Two days later the tension was 35. Four weeks later it had risen to 65 again, and on November 12 the previous operative site was explored. On both sides of the superior rectus muscle the area previously operated on was occupied by a soft, boggy ectatic mass apparently composed of new fibrous tissue formations, with no choroid visible. The masses were spongy and protuberant. A type C sclerotomy was done temporal to the site of the previous sclerectomy. Vitreous was lost, and the eye became too soft for tonometric measurement. On November 20 the tension was again 47, although the eye was no longer painful. The patient was discharged back to the medical ward on December 13 with a tension of 35 in the eye operated on. He did not return for follow-up observation.

CASE 3.—L. K., a white man aged 72, was admitted on Oct. 9, 1940 complaining of severe pain in the right eye. A diagnosis of absolute glaucoma was made. There was no response to conservative measures, and on October 17 a type B sclerotomy was performed on each side of the superior rectus muscle. Vitreous was lost, and the eye became soft. Within three days the tension had risen to 29, and on October 25 it was 56 again. On November 7 the site of the previous operation was explored, and the incisions were found to be gaping slightly, with the choroidal layer barely visible under cover of a thin grayish layer of new fibrous tissue. The surrounding sclera was thin and ectatic. A bilateral type B sclerotomy was again done lateral to the previous incisions, and the tension dropped to 29. Within two days the tension was back to 56 again. Although the pain had eased, the tension remained around this level until December 5, when the patient was signed out. He was not seen again.

CASE 4.—B. B., a white woman aged 72, was admitted on Nov. 7, 1940 with a diagnosis of aphakia and absolute glaucoma (probably postoperative) in the right eye. On November 14 a type B sclerotomy was done bilateral to the right superior rectus muscle. The tension immediately dropped from 56 to 19; it stayed down for one week and then gradually rose again until, three weeks after the operation, it was 65. There was no pain. Enucleation was suggested and refused. The patient left the hospital, to be seen no more.

CASE 5.—N. D., a white woman aged 72, was admitted on Dec. 7, 1940 with a diagnosis of acute glaucoma in the left eye. The tension was 90 +, and the vision was 20/200. The response to use of miotics and other conservative measures was only slight. The following day a temporal type B sclerectomy was done in which an oval of sclera 5 by 10 mm. was excised. The tension dropped to 35. The drop in tension was immediately followed by a broad basal iridectomy. The pain and tension immediately subsided, and the patient was discharged on December 17 with a tension of 17 and vision of 20/100 in the eye operated on. Two weeks later the vision was 20/50. The eye has since been painless and normal in tension. On December 9 the patient was readmitted with the diagnosis of senile cataract in the left eye; the tension was 22. An intracapsular cataract extraction was performed on December 11, and the postoperative course was uneventful.

CASE 6.—H. S., a white man aged 56, was admitted on Dec. 13, 1940 with the diagnosis of an acute exacerbation of chronic glaucoma in the left eye. The tension was 75, and the vision was 12/200. At first the tension responded to miotics, but it soon rose again; on December 22 a temporal type B sclerotomy was done, and the tension dropped from 65 to 22. The drop was immediately followed by a sclerocorneal trephining. The patient was discharged to the outpatient department on December 30 for follow-up observation. He was seen from time to time until Nov. 24, 1941. The tension did not rise above 25 in the affected eye, but the vision dropped to light perception.

CASE 7.—B. P., a white man aged 82, was admitted on Nov. 27, 1940 with a diagnosis of old uveitis and secondary glaucoma in the left eye. The vision was 20/70, and the tension was 56. With the use of miotics, massage and retrobulbar injections the tension was controlled, and the patient was discharged on December 5 with a tension of 17 in the affected eye. He was readmitted on Jan. 2, 1941 with a tension of 65 in the left eye, which this time did not yield to conservative measures. On the following day a temporal sclerectomy of type B was done, and the tension dropped to 35. The drop was followed by a modified Lagrange operation. During the following ten day stay in the hospital, tension and pain were controlled. When the patient was discharged on January 13 the tension in the affected eye was 13 and the vision was 20/200. He did not appear in the clinic for follow-up observation.

CASE 8.—C. A., a white man aged 62, was admitted on Jan. 6, 1941 with the diagnosis of aphakia and (postoperative) absolute glaucoma in the right eye. The tension was 65. Three years prior to admission "he had been operated on in the right eye for a corneal ulcer" at another hospital. Two years before an extracapsular extraction was performed in the same eye. On January 10 a temporal type B sclerectomy was performed in the right eye, and the tension dropped from 65 to 40. The drop was followed by a cyclodialysis. Three days later the tension was 19, and it stayed at this level until discharge on January 31. The tension stayed controlled for the four months during which the patient was under surveillance.

CASE 9.—D. M., a white man aged 67, was admitted on Jan. 10, 1941 with the diagnosis of absolute glaucoma and secondary cataract in the right eye. The tension was 90 +. Two previous operations had been done in the affected eye, a basal iridectomy and a corneoscleral trephining. On January 17 a temporal type B sclerectomy was done, during which vitreous was lost and the eye became soft.

Ten days later the tension again rose to 40, and it fluctuated between 30 and 40 until February 24, when the patient was discharged to the outpatient department. He did not reappear there.

CASE 10.—L. R., a white woman aged 65, was admitted on Feb. 13, 1941 with the diagnosis of old uveitis and absolute glaucoma of the left eye. The tension was 90 +. On the following day a type D sclerotomy was done, and the tension dropped to 54. The drop in tension was followed by a basal iridectomy. The tension fluctuated between 29 and 47, but the pain was controlled by 2 per cent pilocarpine nitrate solution. The patient was last seen on August 9, at which time the tension was 25 and there was no pain in the eye.

CASE 11.—C. T., a white woman aged 51, was admitted on Feb. 27, 1941 with the diagnosis of absolute glaucoma in the right eye. The tension was 76. On the following day a temporal type B sclerectomy was done, and the tension dropped from 76 to 35. The drop in tension was followed by a broad basal iridectomy. Nine days later the tension was 76 again. When the patient was last seen, on November 27, the eye was still hard but not painful. Further surgical intervention was refused.

CASE 12.—T. S., a white man aged 82, was admitted on March 4, 1941 with the diagnosis of absolute glaucoma in the left eye. The tension was 65. The eye was injected and painful and did not respond to conservative measures. On March 10 a type D sclerotomy was done in the upper temporal quadrant, and the tension dropped to 29. The drop in tension was followed by a modified Lagrange filtering operation. The tension was controlled, and the patient was discharged on March 20 with the tension at 19. Thereafter it gradually rose to 35 but has never been higher while controlled with 2 per cent pilocarpine solution. According to the patient there has been practically no pain since the operation.

CASE 13.—J. M., a white man aged 50, was admitted on June 12, 1941 with a diagnosis of secondary glaucoma and cataract. The tension was 80; vision was limited to light projection. Under conservative treatment the tension dropped only to 65. On June 16 a type B sclerectomy was done in the temporal quadrant, and the tension was reduced from 65 to 30. A basal iridectomy was then done. The patient was discharged on June 30 with a tension of 22 in the eye operated on; this was later controlled. When the patient was last seen, on December 3, the tension was 25 in the affected eye. Cataract extraction was deemed inadvisable owing to the poor light projection, especially since the patient was comfortable.

CASE 14.—A. B., a white man aged 67, was admitted on June 16, 1941 with the diagnosis of aphakia and absolute glaucoma in the right eye. The tension was 60. He gave a history of an extracapsular cataract extraction on the right in June 1939, with almost constant pain since. On July 7, 1941 a type D sclerotomy was done, and the tension dropped to 35. The drop in tension was followed by a cyclodialysis. He was discharged on July 16 with a tension of 19 in the eye operated on and with the pain controlled. He did not reappear for follow-up observation.

CASE 15.—A. W., a white woman aged 65, was admitted on July 18, 1941 with the diagnosis of an acute exacerbation of chronic glaucoma. She gave a history of "trouble with the right eye" for the past thirty-five years. The tension was 60 and the vision was 20/100 in the affected eye. On July 21 a type D sclerotomy was done, and the tension dropped from 65 to 40. A modified Lagrange operation

was then done, with total iridectomy. The patient was followed in the period until August 20, during which time the tension was under control and the pain relieved. The vision in the right eye was 20/200 when she was last seen.

RESULTS

Perhaps the most noteworthy result to record is the fact that although 10 of the 15 eyes reported on here presented absolute glaucoma—of which most, if not all, would normally have come to enucleation—not an eye was removed while under observation. This fact is probably due to another unexpected result: the relief of pain noted in almost every case. A third significant fact is that in no case has a delayed infection developed. This despite the removal of rather large segments of sclera in some cases.

On the other hand, it must be noted that the follow-up record is not good, since only 7 of the 15 reported cases have been kept under surveillance to date. This probably compares with the follow-up record of the average municipal hospital in a large city serving a mixed peripatetic population. Of the 7 patients reporting back to the outpatient department, patient 5 has been followed for one year, patient 6 for eleven months, patient 11 for nine months, patient 12 for eight months, patients 10 and 13 for six months and patient 8 for four months. The others were followed for only six weeks or less after operation.

Tension.—With operations 3, 5 and 11 excluded because of loss of vitreous and operation 7 because the drop in tension may have been due in part to a retrobulbar injection given preoperatively, the decrease in tension recorded as the result of simple posterior sclerotomy or sclerectomy varied from 25 to 45 mm. of mercury as recorded with the Schiøtz tonometer. This is an average of 34.6 mm. for thirteen operations, and it does not take into account the fact that the tension was over 90 in several cases and hence too high to be measured accurately. As has already been pointed out, the type of procedure adopted seemed to have no effect on the amount of drop in tension. On the other hand, the higher the tension preoperatively, the greater the diminution obtained on the average.

Any lingering hope which might have been entertained as to the permanence of reduction in tension with these procedures was quickly and thoroughly dispelled by cases 2 and 3, in which three sclerectomies and four sclerotomies, respectively, were done, without permanent effect. It is therefore obvious, from the results listed in table 2, that Robertson,[12] Galezowski[11] and others of the early ophthalmologists were overenthusiastic in their estimate of the value of the posterior sclerotomy or sclerectomy as a weapon for the permanent control of increased intra-

ocular pressure. That a sufficient opening in the inelastic sclera will cause an immediate and appreciable drop in tension has been amply proved (table 1). But it appears to be just as certain that the drop will not remain permanent with this procedure alone. As shown by the results listed in table 2, of the 7 cases which could be followed to the time of writing (cases 5, 6, 8, 10, 11, 12 and 13), the tension has been controlled in 4 (cases 5, 6, 8 and 13). In each of these a filtering operation was also done. Only 1 was a case of absolute glaucoma (case 8). The tension was also controlled in 4 of the remaining 8 cases followed for only a short period of time, and in 3 of these a filtering operation was also done (cases 7, 14 and 15).

Pain.—Much more successful was the control of pain. Even in those cases in which the tension was uncontrolled or in which miotics had to be used postoperatively, pain was either totally abolished or diminished (table 2).

Fortunately or unfortunately this fact has prevented the obtaining of an eye for microscopic study to date. However, inspection in situ of the eye in cases 2 and 3, in which operation was repeated four and three weeks, respectively, after the primary procedure, showed that the sclera where either incision or excision is done becomes filled up with new, weaker tissue, which seems to be less dense and more elastic than normal sclera and which goes on to ectasia owing to continued intraocular pressure. Since there is no cessation of the processes making for the increase in intraocular pressure when the sclera is incised or excised in a glaucomatous eye, the inherent glaucomatous process goes on constantly, tending to convert the newly formed reparative tissue into a bulging cystoid cicatrix. While this tends to modify the tension only slightly or not at all, it does seem to have an effect in relieving pain. Admittedly, if the object is reduction of pain alone, this can be attained more simply by the retrobulbar injection of alcohol or procaine hydrochloride. But when reduction of tension is also desired, simple posterior sclerotomy or sclerectomy seems to be the better procedure and, furthermore, reduction of pain seems to be more permanent.

SUMMARY AND CONCLUSIONS

Fifteen cases of glaucoma are reported in which seventeen single or double simple posterior sclerotomies or sclerectomies were performed. An average drop in tension of 34.6 mm. of mercury was obtained.

The diminution in tension obtained with these procedures alone is not permanent.

Although the work reported here was primarily experimental and done mostly on hopelessly blind eyes, it is suggested that the operations

described may be useful as preliminary to more permanent operations for glaucoma on eyes with potentially useful vision in which the preoperative tension is too high for surgical safety.

Simple posterior sclerotomy and sclerectomy seem to offer a method for reasonable reduction of the tension in hypertensive eyes without sudden loss of intraocular contents and without destruction of choroid or retina.

Furthermore, these procedures also seem to offer the possibility of prolonged relief of pain in eyes with glaucoma of long standing which is resistant to other forms of treatment.

Although in 10 of the 15 cases reported here there was absolute glaucoma, not an eye was enucleated while under observation.

Dr. Edward B. Gresser made the original suggestions for this work, and Dr. Edwin M. Shepherd, former resident, helped in the preparation of the material.

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MODIFICATION OF THE DICKEY OPERATION FOR PTOSIS

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In 1936 Dickey [1] described a new operation for ptosis which, he claimed, offered certain advantages over operations of the Motais type. His indication for the operation was the presence of complete ptosis associated with normal function of the superior rectus muscle. In such cases ptosis operations on the levator muscle by the Blaskovicz method are usually disappointing, and either the Motais or some method of attaching the lid to the frontalis muscle is customarily employed. The disadvantages of operations of the latter type are well known, the chief of these being failure of the lid to follow the cornea in looking down, so that an area of sclera is exposed. Hence these operations should probably be reserved for cases of complete ptosis with no function of the superior rectus muscle.

The chief disadvantage of the Motais operation is the occasional detachment of the superior rectus slip from its new attachment to the tarsus. This may occur even when the superior rectus muscle is approached through a cutaneous incision, as in the method of Shoemaker or Kirby. Kirby [2] described this occurrence in some of his cases and advised that a second operation be performed immediately to secure and reattach the tendon slip. Another disadvantage, due to the small size of the tendon slip, is that it pulls on a small central portion of the upper lid and this often gives a peculiar V shape to the palpebral fissure and the skin fold instead of the normal configuration.

Dickey's operation consisted in passing a strip of fascia lata from the patient beneath the middle third of the superior rectus muscle and suturing it to the anterior surface of the tarsus on both sides. He reported on nine operations in 5 cases, with excellent results except in

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1. Dickey, C. A.: Superior Rectus Fascia-Lata Sling in Correction of Ptosis, Am. J. Ophth. **19**:660, 1936.

2. Kirby, D. B.: Blepharoptosis: Technique of Its Surgical Correction, Surg., Gynec. & Obst. **70**:438, 1940.

1 case, which will be discussed later. Colleagues in San Francisco have told us of other successful results by his method, but apparently no cases have been reported since Dickey's original paper. Spaeth [3] described and illustrated the operation and stated that it produces "the firmest attachment that one can obtain between the superior rectus and the tarsus."

An operation employing a similar idea was described by Trainor.[4] He dissected from the upper edge of the tarsus a strip which was passed under the superior rectus muscle and its cut end sutured back in place. This operation, recommended by Wiener and Alvis [5] as the simplest of all operations for ptosis, has not appealed to us for the reason, advanced by Spaeth, that it forms an undesirable adhesion in the upper cul-de-sac which may interfere with downward movement of the eye. It has also the undesirable feature of burying an epithelial surface beneath the superior rectus muscle.

The operation of Wiener, who sutured the levator to the intact superior rectus muscle, would seem to produce an adhesion to the muscle too far from the lid border to accomplish the desired elevation of the lid in cases of complete ptosis.

Impressed by the surgical principle of Dickey's operation, we first tried it, with the modifications to be described, two years ago and have been encouraged to employ it as the operation of choice in cases of a functionless levator associated with a normally functioning superior rectus muscle.

Dickey exposed the superior rectus muscle through a conjunctival incision and after passing the fascia beneath the middle third of the muscle pulled the ends through an incision in the lid and sutured them to the anterior surface of the tarsus. Our modifications consisted in performing the entire operation through a cutaneous incision without opening the conjunctival sac and in passing the fascia beneath the entire superior rectus muscle. There seemed to be the same advantage in this approach as was offered by the Shoemaker-Kirby modification of the Motais procedure. Use of the entire superior rectus muscle, aside from being simpler, seemed to provide a firmer pulley for the fascial slip, and it was found that it did not interfere with the function of a healthy superior rectus muscle, which was traumatized less by this procedure than by the isolation of its middle third. In case 1 the fascia

3. Spaeth, E. B.: The Principles and Practice of Ophthalmic Surgery, Philadelphia, Lea & Febiger, 1939, p. 344.

4. Trainor, M. E.: Operation for Lid Ptosis, Tr. Sect. Ophth., A. M. A., 1935, p. 93.

5. Wiener, M., and Alvis, B. Y.: Surgery of the Eye, Philadelphia, W. B. Saunders Company, 1939, p. 342.

was passed beneath the middle third on the left side and beneath the whole muscle on the right side, with a slightly better effect on the right side (fig. 2 *B*).

The steps in the operation, as performed on seven lids of 5 patients, were as follows (fig. 1):

1. General anesthesia is employed, all patients to date having been children.

2. A fascial strip 8 mm. wide by 4 or 8 cm. long, the exact size depending on whether the operation is to be performed on one or on

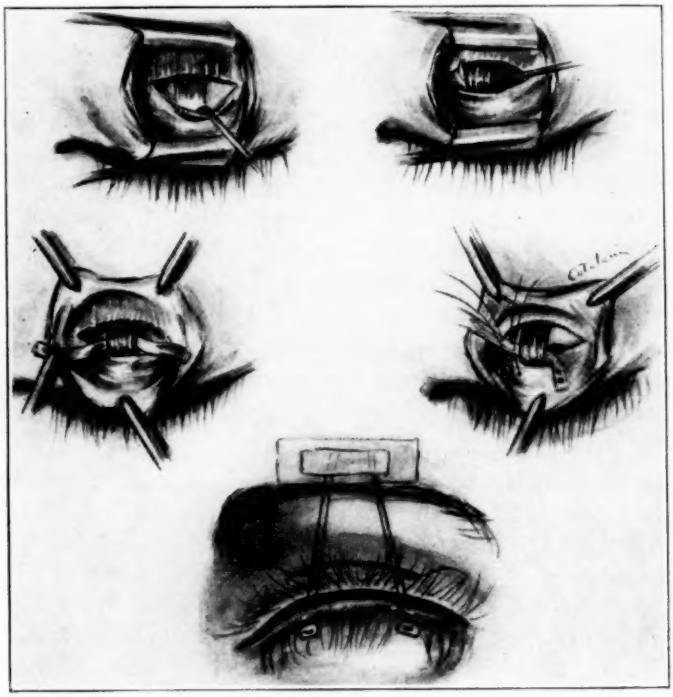

Fig. 1.—Technic of the modified Dickey operation.

both lids, is removed. Use of a fascial stripper allows a short cutaneous incision. This part of the operation may well be performed by an associate or a general surgeon while the ocular structures are being exposed.

3. A horizontal incision is made through the skin and the orbicularis muscles 8 mm. above the lid border. The incision is 20 to 25 mm. long.

4. A dissection is done beneath the orbicularis muscle downward to expose the tarsus and upward to expose the levator tendon.

5. An incision is made through the levator to expose Tenon's capsule as it covers the superior rectus muscle. The dissection and incision must be carried far enough back to miss the upper cul-de-sac. It is of advantage to have the conjunctiva held forward by a tension suture placed in the cul-de-sac, so as to avoid buttonholing it.

6. Two muscle hooks are placed beneath the superior rectus muscle after small nicks are made in Tenon's capsule at each side. The strip of fascia lata, folded with the muscular surfaces together, is passed beneath the superior rectus muscle. This is best done by a suture on a large needle, which is placed through one end of the folded fascia and passed beneath the muscle with the blunt end foremost, the fascia being drawn through by the thread. A clamp or suture is placed on the other end to prevent it from slipping through.

7. The sutures are placed in the tarsus almost as described by Dickey. With double-armed no. 0.009 white silk or linen suture material, a firm bite is first taken in the anterior surface of the tarsus at the junction of the middle and outer thirds and 5 mm. from the lid border. Both needles are then passed through the fascial strip and tied firmly. A second bite in the tarsus is made just below the first bite and the suture passed through the fascia and tied, so as to reenforce the first suture. A bite is now placed at the junction of the middle and inner thirds of the tarsus and 5 mm. from the lid border. The place where the needles of this suture pass through the fascial strip will determine the effect of the operation and should be carefully estimated. The fascial strip is grasped at a point which, when approximated to the tarsal bite, will tighten the strip so as to raise the lid the desired amount. Dickey advised placing the lid border 3 mm. below the limbus, but our experience indicates that a position 1 mm. below the limbus does not give too great a final effect. Unless slight ptosis is present in the other lid, it seems exceedingly unlikely that an overeffect can be obtained. The only operation in which the fascia seemed to slip from its attachment was that in case 3, in which the loop was not made sufficiently tight. After the needles are placed through the fascia at the estimated point, one turn is made in the knot and the effect is examined. If too small or too great an effect is produced, the position of the sutures in the fascia is changed and the sutures tied. The second suture is placed close to and below the first suture, as on the outer side. The loose ends of fascia are cut off close to the second suture.

8. The skin is closed by a single untied subcuticular suture, the fascial sutures being buried. It is not necessary to close the orbicularis muscle separately. It is not removed, as advised by Wiener, but allowed to fall together over the fascial strip.

9. Protection of the cornea is secured by passing two double-armed sutures through the lower lid and out through the lid border. The

sutures are fastened to the brow on adhesive tape so as to pull the lower lid up to the new level of the upper lid, a procedure described by Friedenwald.[6]

10. The sutures in the lower lid may be relaxed by removal of the tape for dressings, which are renewed every second day. They are usually removed after six days, when the subcuticular suture is also removed.

In cases of bilateral ptosis operation is performed on both lids in one session to avoid a repetition of the removal of fascia. Dickey obtained an unsatisfactory result when he attempted to preserve the fascia in alcohol and employ it six weeks later, so that it seems important to employ fascia freshly obtained from the patient. With a pressure bandage of the leg it is not necessary to keep the patient in bed more

Fig. 2 (case 1).—Bilateral ptosis. *A*, before operation. *B*, one year after operation.

than four to five days. Part of the sutures in the leg are removed on the sixth and the remainder on the eighth day, a firm bandage being worn, however, for two weeks.

The results in our small series have been as follows:

Case 1.—M. C., a boy of 5 years, showed complete bilateral ptosis (fig. 2 *A*). Although both superior rectus muscles had appeared to function normally, the right muscle was found at operation to be decidedly lacking in tone. Hence the fascial strip was placed under the entire muscle on this side and under the middle third of the muscle on the left side. The result was satisfactory on both sides (fig. 2 *B*), being slightly better on the side where the whole superior rectus muscle was employed. Since no interference with vertical muscle balance was produced by employing the entire muscle, this was done in subsequent operations.

6. Friedenwald, J. S.: Personal communication to the authors.

CASE 2.—J. D., a girl of 6 years, showed complete ptosis on the right side with a normal superior rectus muscle (fig. 3 *A*). The result is shown in figure 3 *B*. Two months after operation a small nodule developed over the fascial implant. When this was incised a few drops of serum escaped and the nodule disappeared.

CASE 3.—W. S., a boy aged 5 years, showed complete ptosis on the left side. Since the right palpebral fissure was not wide, we were cautious about pulling up the fascial slip. The result was an improvement, but the effect was insufficient. A Machek operation was performed five months later, at which time the site of the implant was exposed. The fascia had apparently slipped away from its tarsal attachment. The result of the second operation is shown in figure 4.

CASE 4.—V. T., a girl aged 1 year, showed complete ptosis and pronounced epicanthus. The action of the superior rectus muscles could not be determined,

Fig. 3 (case 2).—Complete ptosis on the right side. *A*, before operation. *B*, five months after operation.

Fig. 4 (case 3).—Result (three months after operation) of a Dickey operation and a later Machek operation for complete ptosis on the left side.

owing to the age of the patient. Operation was performed because of the extreme backward tipping of the head which was necessary. The result was an improvement, but the effect on the left side was insufficient and a later Machek operation was advised. It seems likely that the superior rectus muscles were not normal in this case.

CASE 5.—S. B., a girl aged 6 years, showed partial ptosis on the right side with some deficiency in the superior rectus muscle and a moderate convergent strabismus. A Blaskovicz operation was first performed, increasing the size of the fissure from 4 to 7.5 mm. Four months later advancement of the superior rectus and recession of the internal rectus muscle was performed. This improved the position of the eye, but as a result more of the cornea was covered by the lid. Hence ten months later it was decided to try a Dickey operation, employing the advanced superior rectus muscle in spite of the obvious drawbacks of such a

procedure, which have been pointed out by White.[7] The effect on the lid was satisfactory. The function of the advanced muscle was, in fact, somewhat interfered with, but the position of the eye was still much better than at first. Diplopia was not a factor, as no binocular vision was present. Some trouble was caused in this case by a granuloma which developed over the fascial slip, requiring incision and cauterization on two occasions. Staphylococci were grown from the material. The incident is mentioned to emphasize the importance of extreme care in cleansing the skin and in asepsis when implanting fascia lata.

SUMMARY

1. The principle of the Dickey operation is such as to recommend it in cases of complete ptosis with normal function of the superior rectus muscle. Other operations are to be preferred when the superior rectus muscle is not normal.

2. Operation entirely through a cutaneous incision seems to eliminate certain disadvantages of the operation as described by Dickey.

3. Passage of the fascial slip under the entire superior rectus muscle is simpler than utilization of the middle third and seems to have no disadvantages.

4. Both functional and cosmetic results are good in suitable cases. A wide palpebral fissure may be secured in cases of extreme ptosis. The lid follows the eye well in vertical movements. The shape of the fissure approximates the normal elliptical one, and the lid fold is usually satisfactory.

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7. White, J. W.: Paralysis of the Superior Rectus and the Inferior Oblique Muscle of the Same Eye, Arch. Ophth. **27**:366 (Feb.) 1942.

CYCLIC OCULOMOTOR PARALYSIS (SPASMUS MOBILIS OCULOMOTORIUS)

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The syndrome of spastic and paretic phases of a cyclic nature in oculomotor paralysis was first describde by Rampoldi in 1884. The first systematic description, however, was published in 1901 by Axenfeld and Schuerenberg, who in addition named the condition. The literature up to 1913 was reviewed by von Hippel, and 18 cases were found. In 1939 Bielschowsky [1] stated that of the 32 cases in the literature he had observed 10.

REPORT OF A CASE

J. R., a man aged 32, was first seen in the early part of 1941 in the clinic of Dr. Willis Knighton at the New York Eye and Ear Infirmary. His past history disclosed that he had had chronic purulent otitis media on the right side for twenty years. At the present time there was no discharge. His ophthalmologic history began at the age of 6 months, when his mother first noticed a drooping of his left lid. This together with an immobility of the eyeball remained the same until 1929, when, at the age of 20, he was unsuccessfully operated on for ptosis. The next year divergent strabismus of his left eye was corrected by a resection of the left medial rectus muscle. The following year a resection of the levator muscle was done, and this too did not improve the appearance of the ptosis. Soon after the last operation the cyclic oculomotor phenomenon appeared.

At the time of examination the patient presented ptosis of the left eye and complete third nerve paralysis of the muscles of this eye; there was little function in the lateral rectus muscle, and the fourth nerve was functioning, as evidenced by internal rotation of the globe when the patient attempted to look in the field of the superior oblique muscle. The left pupil was dilated to a diameter of 8 mm. and reacted sluggishly to light, direct and consensual, but not to accommodation. Bell's phenomenon was not observed. The right eye was normal in all respects, normal vision being obtained after correction of a compound myopic astigmatic error.

In observing the left eye, one saw a little twitching arise in the upper lid; gradually this became quicker and livelier, ending with a complete opening of the palpebral fissure. At this moment the pupil contracted to 3 mm., while the size of the normal right pupil remained unchanged. After twenty seconds the left upper lid went down slowly, and at the same time the pupil dilated again. This cycle repeated itself at irregular intervals all day. During sleep the patient had formerly

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From the Department of Neurology, New York University College of Medicine.

1. Bielschowsky, A.: Am. J. Ophth. **22**:493, 1939.

been bothered once or twice a night, when the phenomenon awakened him, but in recent months he had been awakened only about once a week. During the spastic phase his refraction, as observed with the retinoscope, changed, in that he became 2 D. more myopic, this fact showing the association of the ciliary muscle in the spasm (fig. 1).

The general examination, including tests of the blood and spinal fluid, gave negative results. Roentgenograms of the skull, including the sella turcica, optic foramens and sinuses, showed nothing abnormal. The report on electroencephalography was as follows:

"The record exhibited frequent slow potentials over the left frontal region and over the occipital regions bilaterally. These slow potentials would suddenly disappear for a short time and would then reappear at random. They were chiefly on the order of 4 to 6 cycles per second and of moderate to relatively high amplitude. Some 11 to 12 cycles per second were noted over the parietal and occipital regions.

Fig. 1.—*A*, the patient during the relaxation phase, showing ptosis of the left lid. *B*, the patient during the spastic phase, showing retraction of the left upper lid and miotic pupil.

In certain runs of the record, both frontal regions exhibited a random irregular but frequent appearance of 6 cycles per second, waves occurring fairly synchronously from the two sides of the frontal region."

The slow potentials noted in the record apparently were due to muscular spasms associated with the ocular condition. The relatively normal runs of the record which occurred at certain times might indicate that there was actually no abnormal electrocortical activity.

The neurologic examination showed modifications of the deep reflexes, particularly the patellar, which were decreased and highly fatigable. Involuntary twitchings in the right arm and forearm were noted only by recordings on a kymograph (fig. 2). These twitchings consisted in certain movements of nearly microscopic size and of choreiform character with no regularity in rate or rhythm. There was a tendency to sweating, especially of the face and hands. Fibrillary tremors in the tongue as well as white dermography were noted.

In reviewing the reported cases, it was found that the paresis of the oculomotor nerve was congenital in 14 cases and developed during the first year of life in 8, in the fifth year in 2 and in the seventeenth year in 1. In some cases it was reported with certainty, as in our case, that the cyclic phenomenon came on after the paralysis proper of the oculomotor nerve. In 13 of 17 cases there was amblyopia of higher or lesser degree; in half this was a consequence of astigmatism, whereas in the other half the cause was not ascertained. Refraction in the present case showed vision to be 20/20 in the right eye with a correction of —1.00 D. sph. ⊂ — 0.50 D. cyl., axis 180 and 10/200 in the left eye with a correction of —3.50 D. cyl., axis 180. More than 60 per cent of the patients were females. In 3 cases the trochlear nerve on the same side was affected. In 1 case of Bielschowsky's a temporary dysfunction

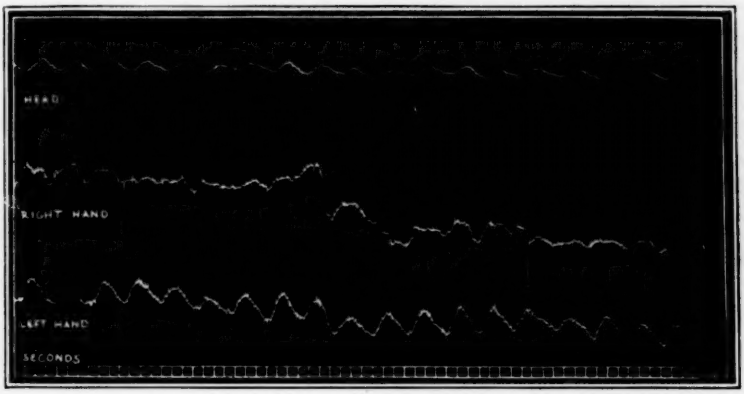

Fig. 2.—Kymographic recordings of the involuntary twitchings in the right arm and forearm.

of the lateral rectus muscle was found. The medial rectus took part in the cyclic phenomena in 12 cases. In all cases some dysfunction of the sphincter pupillae was encountered. The last-mentioned finding was the only one common to all the cases; otherwise all variations of the phenomena occurred. In 10 of 15 cases the ciliary muscle was involved, but in most of the cases the ciliary spasm was less than the pupillary contraction. The levator palpebrae muscle took part in 20 of 28 cases. The muscles of elevation, although paralyzed, took part in the cycle in only 1 case, in which their participation was of minimal degree. In 2 cases rotary nystagmus was observed in the sound eye. In 1 case it was found in both eyes but was dissociated. The cyclic phenomenon was observed many times while the patient was asleep, and it has been known to continue for at least twenty to thirty years. The influence of voluntary ocular movements on the phenomenon has been recorded.

RESULTS OF PUPILLOGRAPHY

1. The pupil in the spastic phase took five seconds to reach its greatest contraction and nine seconds to reach its maximum dilatation. Since the normal time for contraction to light is about 0.45 second, this pupil took ten times as long as normal in its spastic phase. The normal time for redilation after contraction to light is about 0.6 second; so in this case redilation took fifteen times longer than normal (fig. 3).

2. The pupillary unrest in both eyes was exaggerated. It was present in both phases in the left eye and did not disappear when the pupil was dilating but on the contrary was more apparent than when the pupil was contracting.

3. The pupil of the right eye dilated 0.3 mm. on an average when the relaxation phase was occurring in the left eye and contracted 0.6 mm. on an average when the left eye was undergoing the spastic phase.

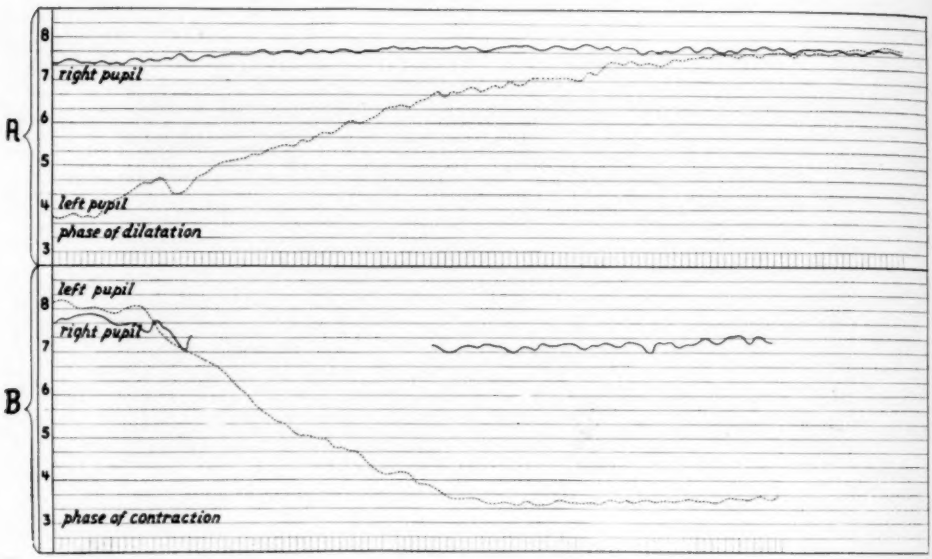

Fig. 3.—Pupillogram demonstrating the pupillary relaxation (*A*) and the pupillary contraction (*B*) phase of the periodic phenomenon.

4. The left eye did not react to near or to far vision during the spastic phase but reacted to the extent of 0.3 mm. in the relaxation phase, while the corresponding reaction in the right eye averaged 1.4 mm. when the left pupil was dilated and 1 mm. when the left pupil was contracted (fig. 4).

5. The reaction to light of the left pupil in mydriasis showed a decrease of its contraction to about half the normal value (that of the opposite eye). The reaction in itself was prompt but showed a prolonged latency period both for contraction and for redilation. Such a reaction is called a tonohaptic reaction[2] (fig. 5). In the stage of spastic contraction the left eye did not show a reaction to light. The con-

2. Lowenstein, O., and Friedman, E. D.: Pupillographic Studies: I, Arch. Ophth., to be published.

sensual reaction of the left pupil was absent during the spastic phase but reappeared when the pupil was about halfway through its mydriatic phase (fig. 6). The reaction which appeared then was not a tonohaptic reaction but a fatigue form of reaction. The latter is found in monkeys with a cut but incompletely regenerated third nerve.

6. The direct reaction of the right pupil was prompt and maximum, but the pupil did not redilate in due time—a sign formerly called neurotonic but now called "an incomplete dilatation block." This syndrome was described (Lowenstein) as a central sympathetic syndrome and is pathologic in man although physiologic in the cat.

7. The reaction to darkness of the right pupil was exaggerated in the dilatation phase, but the second redilatation phase was blocked. The reaction to darkness of the right pupil was relatively independent of the phase of mydriasis or miosis of the left eye. While the stimulated left pupil showed only a small reaction to darkness, the indirect reaction of the right pupil was strong.

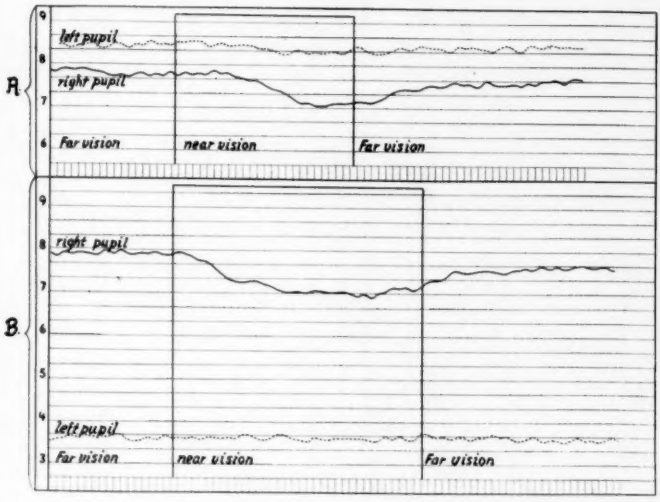

Fig. 4.—*A*, pupillogram of the relaxation phase for near and for far vision, showing a reaction of only 0.3 mm. for the left eye and of 1.4 mm. for the right eye. *B*, pupillogram of the spastic phase for near and for far vision, showing no reaction in the left eye and a reaction of 1.0 mm. in the right eye.

8. Psychosensory dilatation was present but diminished both in the spastic and in the paralytic phase in the left pupil. In the right eye it was exaggerated.

9. The psychosensory restitution phenomenon was absent from the left eye in both the spastic and the paralytic phase but present in the right eye during both phases of the left eye.

10. Physostigmine salicylate produced a contraction in both pupils. If one considers only the paralytic phase the contraction curve of the two sides was approximately parallel. If one considers, however, the spastic phase, the curve of contraction was more sluggish for the left eye than for the right. Physostigmine had no influence on the rhythm of the cyclic phenomena. It is worth noting that a stage was observed in which about twenty-five minutes after the instillation of physostig-

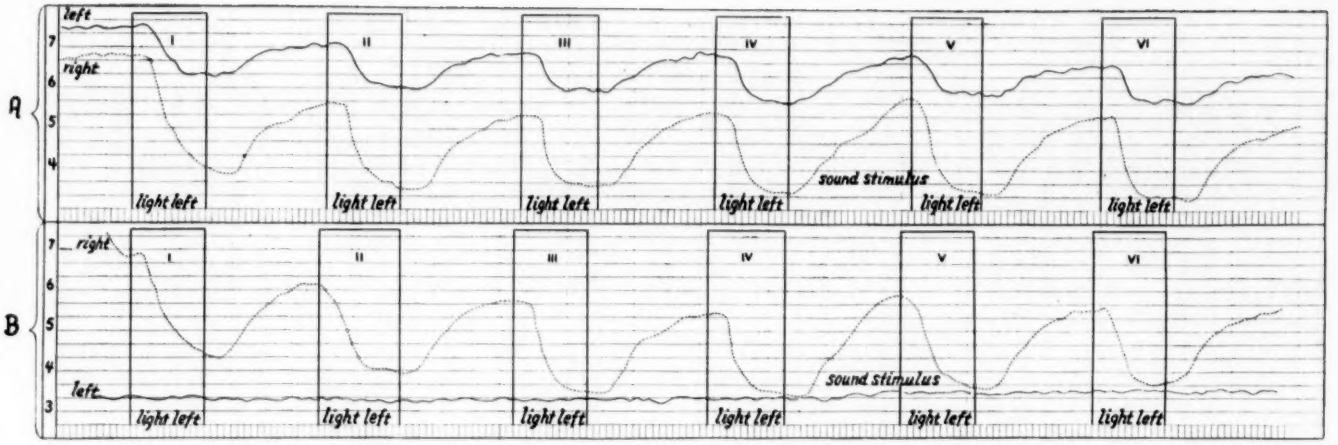

Fig. 5.—*A*, pupillogram of the relaxation phase, showing six reactions to light stimulating the left eye (tonohaptic reactions). There is no psychosensory dilation of the left pupil but good psychosensory dilation of the right pupil. The psychosensory restitution phenomenon is limited to the latency period of the left pupil but is good for the right pupil. *B*, pupillogram of the spastic phase, showing an absence of the reaction to light of the stimulated left pupil but a tonohaptic reaction of the consensually reacting right pupil beginning at the third stimulation. There is slight psychosensory dilation of the left pupil but no psychosensory restitution. There are good psychosensory dilation and good psychosensory restitution of the right pupil.

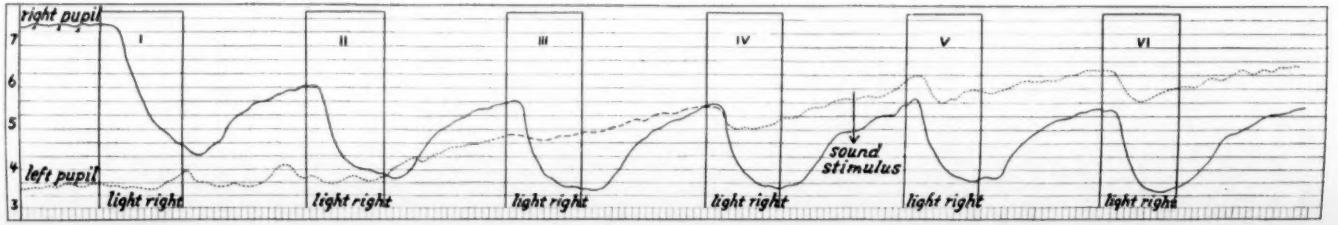

Fig. 6.—Relaxation movement of the left pupil with the right pupil stimulated by light six times. The consensual reactions of the left pupil begin only on the fourth stimulation, when the pupil is halfway dilated. A fatigue type of reaction to the fifth stimulation is shown.

mine salicylate the left pupil reacted better to light in both phases than it did before the instillation. In the relaxation stage, for instance, its diameter was 4.66 mm. and the reaction to light produced a contraction to 1.4 mm.

11. Epinephrine hydrochloride was without influence both in the spastic and in the paralytic phase of the phenomena.

12. Cocaine hydrochloride dilated the left pupil in the spastic and in the paretic phase, so that in both phases the pupils were finally almost equal, but in each phase the relaxed left pupil stayed larger than the right pupil—the spastic left pupil smaller than the right pupil. The rhythmic phenomenon was influenced by cocaine in that there seemed to be a slight tendency to shortening of both the spastic and the relaxation phase.

of the left pupil begin only on the fourth stimulation, when the pupil is halfway dilated. A fatigue type of reaction to the fifth stimulation is shown.

COMMENT

There are two questions worth considering: First, what was the localization of the disturbance and, second, how does one account for the cyclic nature and for the group of muscles involved?

In answer to the first question, Behr[3] and Bielschowsky[4] agreed that this type of lesion is a central lesion. Behr reached this conclusion after finding the Piltz-Westphal lid closure reflex present in 2 cases, an observation suggesting to him that the nucleus of the sphincter and its efferent pathways were intact. Bielschowsky disagreed with Lipschitz' theory that part of the third nerve fibers which might have regenerated had come to the wrong pathways, because he noted that when this does occur there is no refractory interval long enough to be perceived by the naked eye on voluntary movements.

From the pupillographic studies made certain deductions as to localization are possible. We noted that the pupil did not react to light or to near vision in the spastic phase but did so when physostigmine salicylate was instilled in the conjunctival sac. This is interpreted as indicating a diminution of free acetylcholine in the periphery when physostigmine was not present to enhance its action. It was not due to a peripheral lesion, as the inclusion of both the lid and the pupil in the cyclic phenomenon confirmed the integrity of the connection between the nucleus and the periphery as well as the fact that the reaction to light both direct and consensual persisted in the relaxation phase although diminished. We have to assume that the cyclic contraction used up the available acetylcholine, for when the rhythmic contraction receded the pupil reacted to light only after reaching a diameter of 5.3 mm. The degree of contraction to light depended on the lessening of the spasm of the pupil, but even at the maximum of the spasm it could be increased by instillation of physostigmine salicylate.

The reaction to light that we could elicit in the nonspastic phases was of tonohaptic type. Such reactions are generally due to lesions in

3. Behr, C., in Graefe, A., and Saemisch, E. T.: Handbuch der gesamten Augenheilkunde, Berlin, Julius Springer, 1924, vol. 2.

4. Bielschowsky, A.: Arch. f. Ophth. **121**:659, 1929.

the sympathetic centers of the diencephalon or its pathways to the midbrain. The tonohaptic reaction was not present in the normal right eye in the present case. Its absence may be explained if we suppose the interruption of communication between the diencephalon center and the third nerve nucleus to be near the third nerve nucleus itself. This supposition is backed by animal experimentation. If the superior cervical ganglion of a cat is removed, the psychosensory dilatation of the pupil is diminished to about one third of its original value. In a human being under the same circumstances, one finds not only a diminution in the ciliospinal dilatation but a diminution of the psychosensory restitution phenomenon. In cats the latter findings may be fully present. From this fact it is concluded that the psychosensory restitution phenomenon has a peripheral component running over the peripheral sympathetic nerve and a central component which must be considered as a brain reflex and which influences the third nerve center directly by means of a central connection. One of us (Lowenstein) feels that the so-called inhibition center of the third nerve is identical with the sympathetic centers in the diencephalon and the so-called inhibition pathways identical with the ways by which the brain reflex is effective on the third nerve nucleus.

It is known that in man lesions of the diencephalon itself not only produce tonohaptic reactions but suppress the psychosensory restitution phenomenon; however, the phenomena observed in association with these disturbances are generally bilateral. The phenomenon in the present case being unilateral, it is probable that in this case, in which both a unilateral tonohaptic reaction (which was due to a supranuclear lesion —close to the nucleus) and a unilateral diminished reaction to light (which was due to partial destruction of the nucleus itself) were found, we were dealing with a partial destruction of the third nerve nucleus and its supranuclear connections to the so-called inhibition center.

The pupillographic studies led to practically the same localization as that of Behr and Bielschowsky.

The second question to be considered is: What was the mechanism of the automatism and its rhythm and how did the particular association of muscles involved in the phenomenon come about?

Behr and Bielschowsky referred to the blood supply of the sphincter area. They emphasized that in all cases observed the sphincter iridis and the levator palpebrae muscle were constantly affected when two muscles or more were involved. The nuclei of the third nerve branches destined for them are in close proximity in the anterior pole of the third nerve nucleus. The nuclei of those extraocular muscles that were paralyzed and did not take part in the cyclic phenomenon rhythmically are situated in the middle and posterior parts of the nucleus. According to the researches of Bernheimer, each of those areas (anterior and

posterior) has a different blood supply. The anterior pole is supplied by branches from the posterior communicating artery coming from the internal carotid artery. The posterior pole supply comes from branches of the posterior cerebral artery, which in turn comes from the basilar artery. These differences make it possible, according to both Behr and Bielschowsky, that the cells of the anterior nuclear area are spared from complete degeneration, while those of the posterior area degenerate completely.

> That leads to an aplasia of the corticonuclear pathways and the reflex contact neurons. Therefore the anterior nuclei can no longer be stimulated either voluntarily or reflexly, but as the nuclei are still preserved, as are the centripetal pathways, the possibility exists that by means of a normal blood supply accumulation of energy develops in the ganglion cells. This energy being discharged in rhythmic fashion to the periphery, a process develops comparable to the athetotic movements of spastic paretic extremities. (Behr.[3])

Bielschowsky agreed with Behr in all essential points but stated the belief that one is not justified in referring to the close relationship of the levator palpebrae and the sphincter pupillae muscle in the anterior pole, as both anatomically and physiologically the nucleus of the levator palpebrae has a closer relationship to the nuclei of the elevators of the eyeball than to the sphincter. However, the elevators with 1 exception in 32 cases did not take part in the cyclic phenomenon in a rhythmic way. Bielschowsky referred to the researches of Lenz, who found that in the nucleus medialis a larger unpaired artery exists, having developed from two convergent branches from both arteries of the oculomotor nuclei. This unpaired artery gives off branches to both sides, but there exist rare variations, cases in which the artery is absent, and it is exclusively in these cases according to Bielschowsky that cyclic oculomotor phenomena can occur. He stated the belief that in all other instances cyclic oculomotor phenomena as unilateral disturbances are impossible.

Bielschowsky further emphasized that the muscles involved in the cycle may be stimulated only indirectly, as the direct corticonuclear connections are interrupted. In innervating a muscle group the blood supply of the nucleus is considered to be increased and reciprocally that of its antagonists decreased. Therefore when the blood supply of the abducens nerve nucleus increases the blood supply of the third nerve nucleus is diminished, but the decrease is followed by an increase as soon as the impulse to the abducens nerve is relaxed. This increased blood supply to the area of the third nerve nucleus increases the ability to react, as the nucleus can be reached only by afferent stimuli (which neither come from the cortex nor run in the physiologic reflex pathways) and by stimuli coming from certain subcortical centers which alone are able to elicit automatic movements.

There seem to be a certain number of objections to these theories. First, the pupil can be stimulated reflexly in the relaxation period both

directly and indirectly by light stimuli and in the spastic phase after instillation of physostigmine salicylate (figs. 5 and 6). Second, when the increasing tonus of an agonist causes a decreasing of the tonus of the antagonist muscle (Sherrington's law of reciprocal innervation) one is dealing with a reaction which at least primarily is of a reflex nature and bound to intact nervous pathways. It is possible and even probable (but not at all proved) that it is related to modifications of the blood supply, but it is improbable that the reflex effect on the nucleus of the antagonist group could be absent while the effect on the respective vessel areas was present as long as the nucleus itself existed.

According to all observations the so-called indirect stimulation of the third nerve nucleus is possible only by strong voluntary efforts continued for at least several seconds. Its success is little if any compared with the degree of the impulses applied, and no reasonable relation to the force and the dimensions of the automatic movements exists. These movements are apparently independent of all external stimuli and all voluntary impulses. According to Bielschowsky they are even present during sleep, as they were in the present case. Statements in the literature suggest that in the reported cases the movements developed either congenitally or in youth and have existed for years and even decades. We have to assume that in such cases the blood supply of the nucleus area has been stabilized to a high degree if we really are dealing with a nonprogressive or a slowly progressive condition. If alternating stages of increased and decreased excitability result from the condition of the blood supply, we have to suppose that they (as in cases of cardiac activity) must occur in really equal time intervals and with absolute regularity. In conditions which have existed for so long and are not in quick evolution the areas get the same quantity of blood in each time interval. Such regularity did not exist in any case described, as contraction phases, dilation phases and intervals were all different in the same case as to duration—not as to intensity.

If we look for automatic movements with unequal time intervals in the organism they may be found not only in the peristaltic movements of the intestines but in the iris itself, with its so-called pupillary unrest.[5] We know that in these movements we are dealing with processes which in different persons may be of different degrees. They keep the border of the iris in constant unrest and are independent of the innervations of the sympathetic nerves and dependent exclusively on the parasympathetic nerves. They persist in sleep, they disappear when there is a lesion of the nucleus of the third nerve and they increase when there is a supranuclear lesion. The normal pupillary unrest exists simultaneously in several portions of the circumference as slow contraction

5. Lowenstein, O.: Monatschr. f. Psychiat. u. Neurol. **66**:126, 1927.

movements go over into slow dilation while the other parts of the circumference either stay quiet or at least partly perform corresponding alternating contraction and dilation movements; thus the picture of peristaltic movement of the iris border develops.

These movements may be diminished or increased in pathologic conditions; for instance, in Argyll Robertson pupils and in pupils with absolute rigidity, pupillary unrest may finally disappear totally, but in the beginning of each condition, especially of the absolute absence of the reflex to light and convergence, it may be increased. Behr described cases in which pupillary unrest had disappeared in most parts of the iris border but persisted in one circumscribed area, and in this place remnants of a partial reaction to light could be found. Such anomalies are to be found more frequently and are more typical in the beginning of absolute rigidity than in the beginning of Argyll Robertson pupil. Cases were observed in which an increasing or a pathologic type of pupillary unrest preceded the development of disturbances of the light reflex by a long time. Behr concluded that in cases of pathologic pupillary unrest one is dealing with symptoms due to stimulations of the nucleus by the lesions. They stop only when the descending degeneration is completed and the light reflex no longer exists. In the present case vermiform sluggish movements of pupillary unrest were more increased in intensity in the left than in the right pupil. If one looks at the ascending and the descending lines by which the pupillary unrest was recorded, one finds that they form an equal angle to the horizontal —that is, they go on with the same velocity as the rhythmic movements of the left pupil itself. This velocity is different from that of the ascending and descending lines in the light reflex of the same pupil. If the recorded movements in the pupillograms are followed the contractions and dilations of the cycle are found to develop from the small movements of the pupillary unrest and are nothing but their continuation with the same degree of speed. It would seem therefore that there must be stimuli of the same nature which by means of the same mechanism produce the different movements, pupillary unrest and the cyclic phenomenon. The irregularity of the cyclic movements as to amplitude, the explanation of which is impossible by the theories of Bielschowsky and Behr, is explained by the nature of the movements of pupillary unrest, which like those of intestinal peristalsis are inherently irregular.

It is a well known fact that each neuron deprived of its central connection increases in irritability. This explains why the small movements of pupillary unrest can be the stimuli which make the hypersensitive center of the iris perform maximum contraction. It also explains how cyclic phenomena can persist in sleep—as pupillary unrest does.

How does the periodicity particular to the cycle develop? The movements of pupillary unrest follow one another with intervals of part

of a second, while the intervals of the cycle amount to several seconds. To explain this fact, we recall a principle stated by Lowenstein.[6] Normal pupillary reflexes (like all vegetative reflexes) fatigue. This exhaustion is characterized by well defined stages which are transitory and gradually progressive. In the stage of exhaustion the pupil is refractory to induced stimuli of the same kind. In degenerations of the nervous centers, fatigue comes on more quickly than normal and the refractory stage following each stimulation increases. In the normal pupil reacting to light stimuli of one second with intervals of three seconds exhaustion appears after sixty stimuli and a refractory stage appears which is longer than three seconds. In disease, for instance syphilis of the central nervous system, a refractory stage of three or more seconds can be observed after the first stimulation.

We were dealing with such a prolongation of the refractory stage in the present case; the refractory stage of the light reflex, for instance, took the whole phase of a cycle, as shown in figure 3. The light reflex could be elicited once more only after the dilating pupil had arrived at a certain degree of dilatation.

The central spreading of the stimulus from the iris nucleus to the nucleus of the levator palpebrae muscle must be explained by the anatomic localization of the processes and not by the blood supply. If one considers the 32 cases reported on in the literature, none of the muscle supplied by the third nerve have been excluded from participation. It is true that the relative frequency of association seems to be different for the different muscles, but, as the total number of observed cases amounts to only 32, the relative frequency may be decisively modified when the number of observed cases increases. This seems the more probable as the neurologic findings suggest a relatively widespread extent of central involvement.

As to the nature of the condition present, we may be dealing with a progressive process, which in the present case, as in a number of others described, developed at least some of its manifestations as the patient grew older.

Whether we are dealing primarily with a degenerative or an inflammatory process cannot be decided, as anatomic observations are lacking.

CONCLUSIONS

1. The oculomotor phenomenon described is characterized by rhythmic alternations (with irregular intervals) of spastic contraction and relaxation of the iris sphincter muscle. The pupillary movements are generally synchronous with those of other muscles supplied by the third

6. Lowenstein, O.: Der psychische Restitutionseffekt, Basel, Benno Schwabe & Co., 1937.

nerve. The levator palpebrae muscle is most frequently involved, but the ciliary body and the extraocular muscles supplied by the third, fourth and sixth nerves may also be involved. In the present case in addition there were a diminution of the deep reflexes and small involuntary movements of the choreatic type in the upper extremity on the opposite side, pointing to a participation of the ipsilateral striopallidum.

2. As to the location of the lesions producing the automatic oculomotor phenomenon, pupillography shows that we are dealing with a partial destruction of the sphincter nucleus combined with supranuclear lesions involving, among others, particularly the connections between the sphincter nucleus and the hypothalamus.

3. The pupillographic picture further shows that there is no paralysis of the sphincter muscle. As the pupillary symptoms are the only ones to be constant in the syndrome in all cases ever reported, we suggest that the syndrome be spoken of no longer as a cyclic oculomotor paralysis but rather as an intermittent spastic third nerve phenomenon with irregular periods (spasmus mobilis oculomotorius).

4. The periodic spastic oculomotor nerve phenomenon is part of a syndrome due to an extensive process which may be heredodegenerative or postinflammatory (postencephalitic) in nature involving the area of the eye muscle nuclei as well as the basal ganglions. It shows the characteristics of a slow progressive extrapyramidal disease.

5. The association of the pupillary phenomenon with different extraocular muscles is explained by the extension of the basic pathologic process in each individual case.

6. The automatic nature of the movements is explained by the particular combination of (*a*) partial destruction of the sphincter nucleus, which produces increased fatigue, combined with (*b*) a supranuclear lesion, which produces an increased irritability of the remaining part of the nucleus. The increased fatigue produces a pathologically prolonged refractory stage; the increased irritability causes small stimuli, normally below the threshold, to become effective. Such stimuli are those of pupillary unrest. They also explain the persistence of the periodic spastic oculomotor phenomenon during sleep.

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BINOCULAR VISION—NORMAL AND ABNORMAL

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The basic problems of retinal binocular stereopsis are centered around the conceptions of correspondence and disparity. Hering[1] unquestionably laid the foundations for a comprehensive understanding of these conceptions. The weakness of his theories lies in their static, geometric character: According to Hering, whether two points are corresponding or disparate depends on whether the distances from the foveas are the same or different; furthermore, a strict relationship exists between the amount of depth and disparity.

This article attempts to demonstrate that a dynamic rather than a static, geometric conception of stereopsis is correct.

It seems appropriate to start the analysis with a simple dynamic phenomenon, well known but rarely if ever utilized for the interpretation of stereoscopic processes.

Figure 1 presents two stereoscopic drawings, one for the left and the other for the right eye.

The two scales seen through the stereoscope appear as a single scale united in the plane of fixation. The lines *a* and *b*, coinciding monocularly with points 2 and 3, respectively, are disparate and appear as a single line behind the plane of fixation. Most interesting is the functional change of projection of the binocular line with respect to the monocular images. The binocularly united lines appear between 2 and 3 of the binocular scale. This means that the line *a* is seen displaced toward the right and the line *b* toward the left. In retinal terms: Each line appears displaced toward the temporal side. The binocular image of *c* and *d*, appearing before the fixation plane, also is seen between 2 and 3; but here the displacement is nasal. This simple demonstration reveals two important facts:

1. Binocular stereopsis is connected with a functional change of projection.

2. Direction of depth and direction of displacement are strictly correlated. Functional displacement nasally signifies a position in front of, and displacement toward the temporal side a position behind, the fixation plane.

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1. Hering, E.: Die Lehre vom binokularen Sehen, Leipzig, Wilhelm Engelmann, 1868.

The question could be raised concerning the correctness of the term "functional displacement." One might question whether one is justified in comparing the projection of a monocular image (half-image) with that of the binocular image. However, it is possible to demonstrate that the functional change of projection occurs actually with each "half-image." For this demonstration a "strobostereoscope" has been used.[2] This apparatus permits the projection of stereoscopic pictures in such a way that the two retinal images appear one after the other.

The strobostereoscopic experiment proves two facts:

1. In sufficiently rapid succession disparate half-images produce a stereoscopic effect concomitant with functional displacement.

2. Functional displacement of each half-image separately can be observed.

How general is the relation between stereopsis and functional displacement? The question can be answered correctly only after an

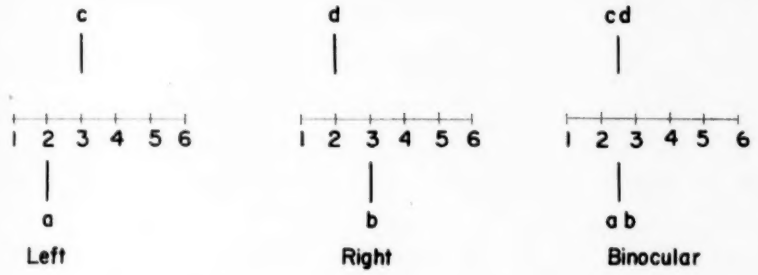

Fig. 1.—Stereoscopic card demonstrating displacement.

analysis of stereopsis in diplopic vision. Both older and more recent experiments have demonstrated that fusion of the half-images is not essential for depth perception.

The interpretations of the relative depth effect in diplopic vision appearing in textbooks on physiology and psychology are frequently given in terms of the difference between crossed and uncrossed double images. It is contended that double images contribute to stereopsis because the difference in relative position is apprehended by the organism as difference in relative depth: The two images if crossed appear before and if uncrossed appear behind the fixation plane. Such an interpretation cannot be reconciled with the facts. Since it is impossible just by looking at the double image to see whether it is crossed or uncrossed, such a relationship per se can hardly be a psychologic basis

2. Werner, H.: Dynamics in Binocular Depth Perception, Psychological Monographs, Columbus, Ohio, Psychological Review Company, 1937, vol. 49, no. 2, p. 51.

for stereopsis. Furthermore, a double image (determined objectively by a definite geometric disparation) may appear at varying distances from the fixation plane; the variation may extend from full appreciation of relative depth to zero. This fact alone would show that interpretations in terms of geometric retinal conditions are incomplete. All indications point to dynamic factors underlying stereoscopic vision of double images.

What are the main facts? Suppose one presents to an observer two parallel lines, one line, F, being the focus line and the other line, A, being to the right side and behind the fixation plane. F appears as a single binocular image, $f_1\, f_2$; A, as a double image, $a_1\, a_2$ (fig. 2).

If the depth effect of the disunited double image $a_1\, a_2$ is of the same nature as the depth effect of any united binocular image, one would expect a displacement of each half-image with respect to its projection seen monocularly. This assumption can be examined by the following method. By screening off the half-image a_2 and introducing it again

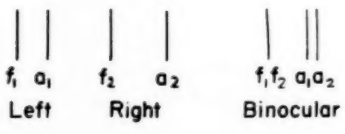

Figure 2

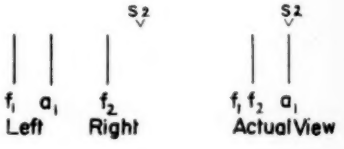

Figure 3

Fig. 2.—Diplopic lines.

Fig. 3.—Nonius method; position of an isolated half-image.

into the visual field, one may determine whether the phenomenal projection of a_1 remains the same or changes, according to the presence or absence of a_2. For this purpose one applies the so-called nonius method: A movable mark, s_2, visible only to the right eye, is shifted into a position exactly above the line a_1 in the binocular field (fig. 3). If a_2 is screened off so that only a_1 is visible, the distance from F appears the same whether Fa_1 is seen by the left eye alone or binocularly. Mark s_2 occupies the position of the line a_1 in the binocular field (the distance $f_2\, s_2$ equals that of $f_1\, a_1$). After introduction of the right-hand image, a_2, into the visual field, a phenomenal displacement of a_1 toward a_2 takes place, indicated by the new position of a_1 with respect to s_2 (fig. 4).

One can measure the amount of displacement by shifting the mark s_2 from its original position till it stands exactly above the new position of a_1. The length of shift signifies the displacement caused by the introduction of the second half-image.

For this experiment the following setup was used:[3] The observer, his head held in a fixed position, looks through an aperture at two black silk threads suspended vertically against a white background. The observer focuses on the left thread. The right-hand thread, being farther behind, appears in uncrossed half-images. The experimental situation is briefly sketched in figure 5.

M and N indicate the two nodal points. F is the left focused thread. A is the right-hand thread appearing in uncrossed half-images a_1 and a_2. Mark S is movable in the plane of fixation. The visual field of the left eye is covered in the upper part by a white screen, L. This screen

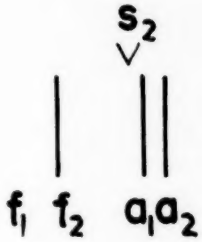

Fig. 4.—Nonius method; position of a double image.

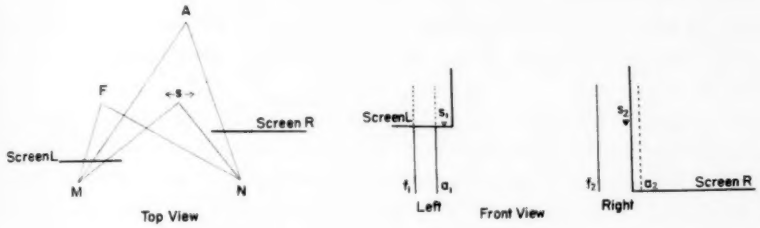

Fig. 5.—Setup of the thread experiment.

covers also the mark S for the left eye. The right eye sees the mark as s_2.

The right-hand half-image a_2 can be covered by removable screen R. The observer controls fixation of F by seeing to it that the upper part of f_2 lies precisely on the extension of f_1.

These experiments with diplopic images lead to the following results: The apparent projection in the binocular field of a half-image, a_1, changes according to whether it is seen isolated or together with the other half-image, a_2. If seen isolated, that is, with exclusion of a_2, the half-image appears generally in the fixation plane and its position in

3. For a detailed description of the experiment and the results see Werner,[2] p. 5.

the binocular field is identical with that held in monocular vision. Under diplopic conditions, however, the projection of the half-image a_1 is shifted in the direction of the other half-image.

I may illustrate by one example: Mark s_2, coinciding with the isolated half-image a_1 in the binocular field, is seen at a distance from the focus indicated by the angle FNS equals 26 minutes 40 seconds. After introduction of the other half-image, s_2 has to be shifted in order to coincide with the now changed position of a_1. The distance of s_2 from the focus is now 34 minutes 47 seconds, indicating a displacement of a_1 of 8 minutes 7 seconds.

It may therefore be concluded that the physiologic processes underlying stereoscopic vision are fundamentally the same whether one is dealing with perfectly united or with diplopic binocular images. The functional change of retinal projection indicates an interaction between the stimulated retinal areas. The direction of the functional displacement defines the direction of relative depth: Nasalward displacement is related to forward temporal displacement to backward direction of depth.

Further experimental evidence, suggesting an interpretation of stereopsis in terms of dynamic-functional events instead of a static, geometric retinal relationship, concerns changes of correspondence.

One may briefly recall "the changes of correspondence" occurring under the abnormal conditions of squint in order to relate such changes later to comparable facts from the experimental psychology of normal vision.

In ordinary binocular vision the image of an object imprinted on the squinting retina deviates from the fovea by an angle equal to the angle of the squint. Therefore, the projection of the object made by the squinting eye should always differ from that of the normal eye. The subject should see the diplopic images at a distance from one another corresponding to the angle of squint. This is in general true at the onset of the squint. But as time passes the projection becomes less incorrect by an angle approaching the angle of deviation; eventually the projection may be correct in spite of the deviation of the eye. This angle of adaptation has been termed the "angle of anomaly."

Figure 6 represents diagrammatically the progressive change in the angle of adaptation in the case of a right convergence.[4] BD indicates the deviation of the eye from the normal direction BO. P is the projection of an object O made originally by the deviating eye. The angle OBD equals the angle PBO; that is, the angle of anomaly is zero. P_1, P_2 and P_x are the projections made by the squinting eye during

4. Worth, C. A.: Worth's Squint, or the Binocular Reflexes and the Treatment of Strabismus, edited by F. B. Chavasse, ed. 7, Philadelphia, P. Blakiston's Son & Co., 1939, p. 399.

successive stages of adaptation. The angle of anomaly expressed in terms of the difference angle *OBD* minus angle *PBO* may increase to a maximum at which P_x becomes identical with *O*. The angle of anomaly equals then the angle of squint. The relative degree of anomaly can be indicated by the perversion quotient $\frac{\text{angle of anomaly}}{\text{angle of squint}}$, ranging from 0 to 1.

Any such change of projection signifies a functional displacement. Since corresponding points are defined by the identity or projection, a change of projection in the squinting eye means a change in correspondence. A new correspondence, a "secondary retinal correspondence," emerges as a consequence of the squint.[5]

Changes of correspondence definitely occur in normal vision. For a simple demonstration the stereoscopic pictures of figure 7 may be used.

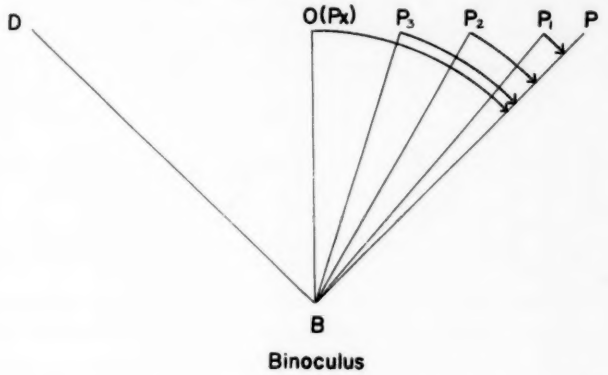

Fig. 6.—Progressive change in the angle of anomaly in a case of right convergence (after Worth [4]). The arrows indicate degrees of anomaly.

If the two rectangles, which differ in width, are stereoscopically united, a plane tilted diagonally should be seen: The left vertical line should stand in front—and the right line in back. In spite of the geometric disparity, some observers see the oblong in a frontal parallel plane, while for others it is only slightly tilted. Defining correspondence by the two characteristics of unification and zero depth, one can assume that for the duration of observation a new, a secondary, correspondence has been established; the points *2* and *4*, which are geometrically disparate (with respect to fixation points *1* and *3*), have become truly

5. Usually, abnormal correspondence is apparent only if both eyes are uncovered. The squinting eye used alone comes straight. It should be further stated that abnormal correspondence attains rarely the precision of normal correspondence. The size of the pseudofoveal area probably never equals in smallness the normal area of fixation.

corresponding points. Consequently, the relationship of other stimulated points becomes transformed with respect to the new frame of reference. Since a secondary correspondence has been established, a new interpretation of disparity, i. e., a secondary disparity, arises. Figure 7 is an illustration. The horizontal lines within the squares are identical in length and therefore are geometrically corresponding. They should therefore be seen as being frontally parallel. Instead, they appear diagonally tilted with the right end in front of, and the left end behind, the fixation plane. In other words, because of the secondary correspondence geometrically corresponding points manifest relative depth; that is, they acquire secondary disparity.[6]

A few remarks may be added concerning monocular diplopia. After operation for squint the patient has to undergo a readjustment toward normal correspondence. One period of readjustment is characterized by a rivalry or balance between abnormal and normal correspondence. The two kinds of correspondence may alternate, or they may exist simultaneously, producing so-called monocular diplopia. One point

Figure 7

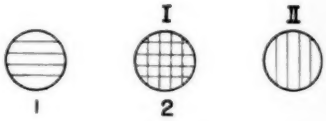

Figure 8

Fig. 7.—Stereoscopic cards demonstrating a change of correspondence.

Fig. 8.—Color mixture of monocularly diplopic images.

of the eye operated on is then related to two points in the other eye and may therefore be projected in two different directions at the same time. Cases of monocular diplopia in which physical or anatomic causes of this phenomenon could be excluded have been described by Tschermak,[7] Javal, Bielschowsky,[8] Koster, Ohm,[9] Zentmayer [10] and others.[11]

6. I have previously shown [2] that changes of correspondence in normal vision can be measured by means of the specially constructed "contrast stereoscope."

7. Tschermak, A.: Ueber anomale Sehrichtungsgemeinschaft, Arch. f. Ophth. **47**:508, 1899.

8. Bielschowsky, A.: Ueber monokuläre Diplopie ohne physikalische Grundlage, Arch. f. Ophth. **46**:143, 1898.

9. Ohm, J.: Klinische Untersuchungen über das Verhalten der anomalen Sehrichtungsgemeinschaft der Netzhäute nach der Schieloperation, Arch. f. Ophth. **67**:439, 1908.

10. Zentmayer, W.: Monocular Diplopia, with Special Reference to That Associated with Cerebral Lesions, Tr. Am. Ophth. Soc. **21**:223, 1923.

11. Monocular diplopia, demonstrated by Best, Fuchs and K. Goldstein (The Organism, New York, American Book Company, 1939, p. 50) in cases of homonymous hemianopia, is probably not of a binocular nature and may therefore be excluded from this discussion.

Bielschowsky has demonstrated the incorrectness of the assumption that monocular double vision is caused by alternation of the two projections. He presented to his patient two circles of different colors so far apart that one image of the one diplopic impression coincided with one image of the other diplopic pair. Color mixture occurred at the points of coincidence of these images (fig. 8).[12]

It can be demonstrated that monocular diplopia may occur also in normal vision under particular experimental conditions. Under such conditions the stereoscopic picture of figure 9 was presented strobostereoscopically so that the two half-images appeared one after the other in rapid succession. When f_1 and f_2 were focused, a and b were usually seen displaced toward each other. Often, however, line a, seen only by the left eye, appeared double. That is, it was seen undisplaced and displaced.

One may attempt to interpret the change of correspondence in normal and in abnormal vision by referring to the dynamic-physiologic

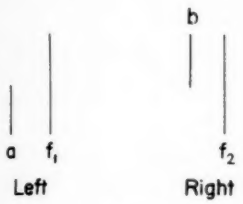

Figure 9

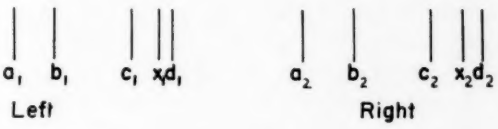

Figure 10

Fig. 9.—Strobostereoscopic card for demonstration of monocular diplopia in normal vision.

Fig. 10.—Stereoscopic card illustrating the relationship of stability and depth effect.

conception of stability or equilibrium. An example may serve to illustrate the relationship between stability of binocular images and depth effect in normal vision. Figure 10 is a stereoscopic card consisting of two groups of lines. Since the disparity of the binocularly united lines a_1 and a_2 and b_1 and b_2 is identical with that of the lines c_1 and c_2 and d_1 and d_2, the stereoscopic effect should be the same. However a and b appear more or less in a frontal parallel plane, whereas c and d are seen turned about at a considerable angle. This difference is caused by the lines x_1 and x_2 lying between c_1 and d_1 and c_2 and d_2 at equal distances from c and c_1.

A further analysis reveals that, whereas the lines b_1 and b_2 stay strictly united, the lines d_1 and d_2 tend to separate into a double image.

12. Hofmann, B.: Die neueren Untersuchungen über das Schielen der Sehenden, Ergebn. d. Physiol. **1**:2, 1902.

In one case the displacement is of great stability. In the other the displacement is unstable; there is a tendency to return to the binocular equilibrium of primary correspondence. In the case of *c* and *d* the tendency to unify retinal points in terms of primary correspondence is present. In the case of *a* and *b* the primary correspondence is, so to speak, forgotten. A new binocular equilibrium has arisen which may serve as a new frame of reference, phenomenally appearing as zero plane.

Two dynamic traits, therefore, distinguish correspondence, namely, binocular unification of retinal points and stability of this binocular unity. Viewed from this dynamic conception, points of primary correspondence are those which, because of neuroanatomic reasons, will most likely insure a high stability during binocular unification. They may therefore serve as a frame of reference for points exhibiting binocular disparity; they constitute the zero which determines the amount of functional displacement and relative depth. If, for any cause, maximal stability is shifted to points hitherto disparate, a new frame of reference, a secondary correspondence, arises.[13]

The shift of correspondence is a consequence of changed stability conditions. These may be short-lived, as in normal vision, or enduring, as in abnormal vision.

What are the retinal conditions which cause geometrically disparate points to acquire high stability? They arise simply from the fact that the binocular unity of disparate points is so strong that the tendency toward unification of anatomically corresponding points is of little or no effect. In normal vision these conditions are produced predominantly by external factors, that is, by the peculiar configuration of the stimuli. If vertical and parallel lines, like the lines a_1 and b_1 and a_2 and b_2 of figure 10, are employed, the tendency toward unification is strong because (1) the disparity is relatively small, (2) the disparity is the same at all points and (3) no other lines either of different disparity or of primary correspondence appear in the field. If the configuration is altered by negating any of the three characteristics it becomes less stable. Such instability arises in the example illustrated by figure 10 from introducing the primarily corresponding lines x_1 and x_2.

The abnormal correspondence of the squint is also caused by a shift of stability conditions. But these conditions are produced predominantly by internal factors. One of these internal factors is the change of direction of gaze in the squinting eye from a normal to an abnormal position. The relationship between direction of gaze and correspondence is clearly demonstrable in a person who can place the squinting eye in the normal position. Such a person exhibits a change from the usual

13. Concerning the general neurologic conception of equilibrium, see Goldstein.[11]

abnormal to normal correspondence concomitant with the change from abnormal to normal gaze (Bielschowsky,[8] Schlodtmann, Ohm[9]). The second factor consists in the shift of the peak of acuity in the squinting eye from the fovea to a pseudofovea (fig. 11).

Retinal inhibition in the foveal area strengthens indirectly the unification of diplopic images. If the foveal deficiency is slight, normal correspondence may still prevail, in spite of diplopia. It must also be remembered that macular inhibition is not always absolute; foveal deficiency may be less apparent if the stimulation of the fovea of the normal eye is less intense. One of Ohm's[9] patients, usually exhibiting abnormal correspondence, returned to normal correspondence as soon as the intensity of the stimulus received by the normal eye was reduced.

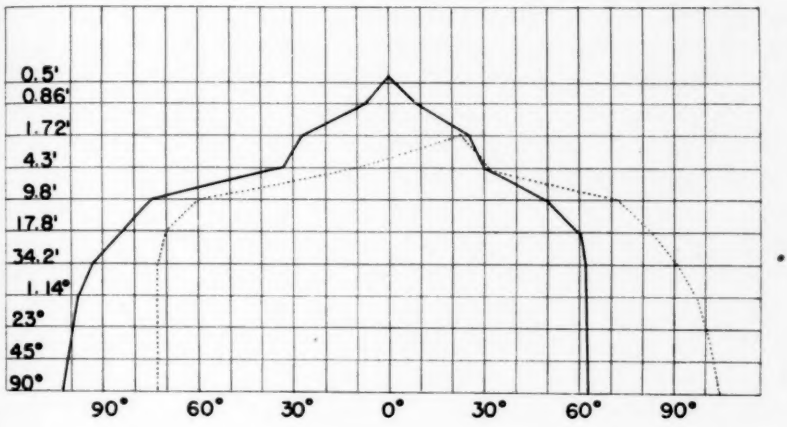

Fig. 11.—Comparison of sensitivity of (continuous line) a normal left eye and (dotted line) a squinting eye (after Worth[4]).

If the foveal deficiency is sufficiently great, the relationship between geometrically corresponding points ceases to possess high stability. The physiologic peak of stability has been shifted in a direction corresponding more or less with the foveal shift. It is not necessary that such shift should lead to a complete adaptation, that is, a complete unification of diplopic images. In paralytic patients, particularly, the change to secondary correspondence is usually less than the angle of squint.[14]

Points of secondary correspondence—provided there is sufficient stability of this relationship—may furnish a new frame of reference. A secondary disparity, that is, stereopsis, may develop on the basis of this new zero plane.

In conclusion, one is justified in assuming that the principles involved in an abnormal retinal relationship can be shown to be effective in

14. Worth,[4] p. 395.

normal binocular vision. Abnormal projection of retinal points, appearing under pathologic conditions, is definitely related to a fundamental characteristic of normal binocular vision, namely, functional displacement of binocularly united or diplopic images. A change of the stereoscopic frame of reference, that is, secondary correspondence and secondary disparity, occurs again permanently in abnormal, and temporarily in normal, vision. Under abnormal circumstances change of correspondence is caused, among other things, by foveal inhibition. In normal vision the temporary loss of primary correspondence is caused by certain stimulus configurations which insure stable unification of disparate images.

Wayne County Training School.

USE-ABUSE THEORY OF CHANGES IN REFRACTION VERSUS BIOLOGIC THEORY

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By the term use-abuse theory I mean for the purposes of this article all the major theories of change in refraction opposed to the biologic theory. The term is used to contrast two views as sharply as possible. Roughly, the use-abuse theory covers the school work, convergence-accommodation, poor lighting and bone formation theories. Near sightedness looms large and dominates the thinking of the school which favors this theory. The biologic theory stakes everything on heredity and growth. For its adherents, near sightedness is a serious state of refraction, but only one type of biologic refraction status. Myopia and its development do not dominate the thinking of the school favoring the biologic theory.

Frankly, both concepts are pure theories. Facts in full support of either are lacking. This article, therefore, only pretends to state the issue more sharply than has been done heretofore in this country and to add a small amount of data on the biologic side.

Cohn, of Breslau, was the first to stress near work as the cause of near sightedness (1866). But since his time certainly the great majority of the leaders in ophthalmology have agreed with him. Outstanding among the early writers were Javal, Landolt, Tscherning, Stilling, and Knies, in Europe; Bishop Harmon, in England, and Hasket Derby and Risley, in this country. Others were early impressed with heredity as a cause, notably Donders, in 1866. Priestley Smith was likewise cautious in accepting the near work view, in 1880, and while Hess, in his Graefe-Saemisch article (1903), found near work generally accepted as the cause of myopia, he stated that this explanation is purely a hypothesis. But it remained for Adolf Steiger to develop fully the biologic theory, in 1913.

The strongest evidence that near work causes near sightedness is that near sightedness increases during the early school years. The increase is from about 1 per cent at the age of 6 to 6 per cent at the age of 11. Some increase continues to the age of 15 or 16. But then it practically stops, despite the fact that for a great many persons the

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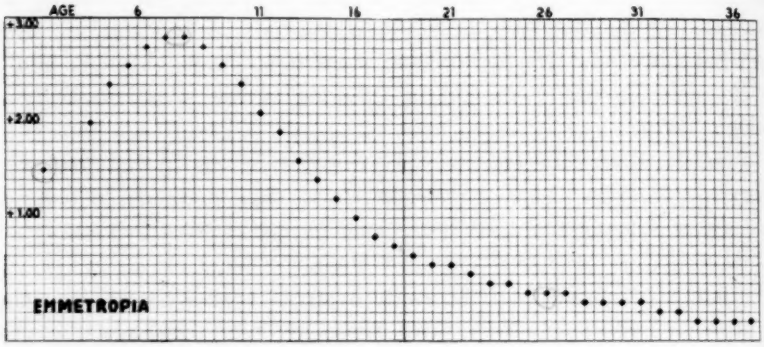
amount of near work, accommodation and convergence continues and indeed increases. The poor lighting theory fares even less well, for with the improvement in lighting from the candlelight days of Abraham Lincoln through the use of kerosene and gas to electricity, near sightedness cannot be said to have decreased any and may well have increased. Stilling's theory, that a relatively low orbit makes the extrinsic muscles press on the bulb unduly in convergence and thereby elongates the eyeball, speaks more for an inherited racial type of skull than it does for use or abuse of the eyes in convergence.

The strongest evidence that normal biologic processes determine or "cause" near sightedness is that much near sightedness is inherited. Whole families are often myopic, the father and mother and all or nearly all the children. And other whole families—father, mother and all or nearly all the children—are hyperopic and remain so. But there



Refraction curve of changes in 1,737 persons, based on the 1.52 D. of hyperopia found in 97 of the eyes before the age of 2.

are many exceptions. Likewise, attempts to trace the effect of this, that or the other thing which affects postnatal growth are beset with difficulty, just as are attempts to trace effects on body height or weight or on intelligence.

My view is that neither use nor abuse has been shown to affect refraction states in any way whatever but that there is still insufficient evidence to warrant full acceptance of the biologic theory. For this reason I should like to present some data of my own in support of the contention that changes in the refraction state conform to some of the rules of biology, in particular that according to which there is a general uniformity of change during the period of growth and a cessation of change afterward.

It is widely held that few eyes are myopic at birth. Yet my associates and I have found that 18 of 97 eyes first studied during atropine

cycloplegia before the age of 2 years averaged 4.32 D. of myopia. The remaining 79 eyes averaged 2.85 D. of hyperopia and the group as a whole 1.52 D. of hyperopia. Great accuracy cannot be expected of so small a group, but, taking this 1.52 D. of hyperopia as a starting point, I have added the average net changes in refraction found in each subsequent year in these patients and in all others—in 1,660 persons, to be exact—who have had two or more atropine tests. From these data

TABLE 1.—*Yearly Refraction Change in 1,737 Persons Retested with Atropine Cycloplegia*

Year of Life	No. of Computations	Net Average Change, D.	Type of Change *	Year of Life	No. of Computations	Net Average Change, D.	Type of Change
1	4	0.77	Decrease	28	168	0.04	Increase
2	86	0.43	Decrease	29	163	0.02	Increase
3	236	0.40	Decrease	30	159	0.02	Increase
4	329	0.28	Decrease	31	158	0.05	Increase
5	437	0.20	Decrease	32	94	0.02	Increase
6	552	0.09	Decrease	33	93	0.09	Increase
7	612	0.02	Increase	34	96	0.02	Increase
8	647	0.06	Increase	35	81	0.04	Decrease
9	751	0.21	Increase	36	93	0.02	Increase
10	812	0.21	Increase	37	82	0.01	Decrease
11	787	0.25	Increase	38	77	0.09	Decrease
12	824	0.26	Increase	39	68	0.04	Decrease
13	784	0.27	Increase	40	67	0.01	Decrease
14	730	0.27	Increase	41	48	0.02	Decrease
15	628	0.21	Increase	42	42	0.11	Decrease
16	524	0.21	Increase	43	39	0.009	Decrease
17	534	0.12	Increase	44	20	0.02	Increase
18	438	0.12	Increase	45	20	0.02	Increase
19	352	0.10	Increase	46	7	0.04	Increase
20	348	0.07	Increase	47	12	0.008	Increase
21	315	0.06	Increase	48	11	0.007	Increase
22	291	0.08	Increase	49	11	0.08	Decrease
23	278	0.07	Increase	50	5	0.05	Increase
24	262	0.05	Increase	51	3	0.03	Increase
25	204	0.05	Increase	52 to 54, incl. no observations			
26	180	0.04	Increase	55 to 74, incl., 1 case (38 computations)			
27	162	0.02	Increase				
Total					13,762	0.09	Increase

* A decrease means more hyperopia or less myopia; an increase, less hyperopia or more myopia.

I have plotted a curve which postulates the average net balance between increases and decreases for the years 2 to 36 inclusive, as shown in the accompanying chart.

Details for each year are given in table 1. The material comprises the eyes of 1,737 persons. For the first and second year the total is less than one hundred computations and so is hardly reliable, but for the ages 3 to 25, when heredity and growth are presumably most effective, the computations number from two hundred to eight hundred for each year and should permit reliable deductions.

These data show a steady, regularly graduated and progressive decrease of refraction (increase of hyperopia or decrease of myopia) for each year up to the end of the sixth. The amounts of decrease get less each year and are 0.43, 0.40, 0.28, 0.20 and 0.09 D., respectively. An increase in the net yearly average change supervenes from the seventh to the fourteenth year. The amount of increase gets larger with each year, viz. 0.02, 0.06, 0.21, 0.21, 0.25, 0.26, 0.27 and 0.27 D. The increase in refraction continues through the fifteenth and sixteenth years at 0.21 D. The seventeenth, eighteenth and nineteenth years each also show an increase. The amounts are, respectively, 0.12, 0.12 and 0.10 D.—barely half the change found at 15 and 16. From 20 to 32 there is almost no change; the slight increase in refraction (move toward the myopic side) varies between only 0.07 and 0.02 D. per year. An irregularity in increase and decrease occurs between 33 and 36 inclusive. From 37 to 43 my figures show a slight decrease, nearly

TABLE 2.—*Summary of Changes in Six Age Groups*

Age, Years	Number of Computations	Amount of Change, D.	Type of Change
1- 6	1,644	0.22	Decrease
7-16	7,099	0.20	Increase
17-19	1,324	0.11	Increase
20-34	2,971	0.05	Increase
35-43	597	0.003	Decrease
44-51	89	0.004	Increase
1,737 cases:	13,762	0.09	Increase

always less than 0.05 D. per year. After 43 there are insignificant increases. It will be noted, however, that after 31 the computations run less than one hundred per year, and this number is too small for reliability. The data for ages beyond 43 are based on even smaller numbers. Patients are loath to submit to a second atropinization at this time of life.

The "atropine material" of the ophthalmologic department at Albert Merritt Billings Hospital, University of Chicago Clinics, has naturally grown through the years, and various aspects of it have been the subject of previous studies by other members of the staff and myself.[1]

My 1938 report was along much the same lines as this one, but covered only 1,203 persons. A decrease of refraction (increase of

1. Brown, E. V. L., and Kronfeld, P. C.: Refractive Curves in the U. S. A. with Special Reference to Changes in the First Two Decades, Compt. rend. Concilium Ophth. **13**:87, 1929. Kronfeld, P. C., and Devney, C.: Ein Beitrag zur Kenntnis der Refraktionskurve, Arch. f. Ophth. **126**:487, 1931. Bothman, L.:

hyperopia) went over into an increase of refraction at the end of the seventh year instead of at the end of the sixth year, as in the present series. The rate of increase of refraction after the age of 20 was 0.04 D. per year in the 1938 data and 0.05 D. per year in the present data.

Table 2 summarizes the changes in six age groups (1 to 6, 7 to 16, 17 to 19, 20 to 34, 35 to 43 and 44 to 51). The 1 to 6 year group shows a definite decrease of refraction (increase of hyperopia), but each of the next three groups shows an increase, this increase lessening in amount in each group.

SUMMARY AND CONCLUSIONS

The use-abuse theory of changes in refraction is based mainly on the known increase in near sightedness during the early school years. The biologic theory is based mainly on the marked inheritance of near sightedness. Neither theory has enough evidence to warrant its full acceptance.

I have studied the refraction changes in the eyes of 1,737 persons under repeated atropinizations, one or more years apart; have computed the net average change in refraction (for each eye separately, in comparison with the ståtus found in the same eye under previous atropinization or atropinizations) throughout the years it was observed, and have plotted a curve showing the net average change found in each year.

Of 97 eyes, 18 were found to be myopic before the age of 2, but the net average refraction was 1.52 D. of hyperopia. The curve found is even, regular and uniform. Each year shows a steady increase in net hyperopia up to the end of the sixth year of life. A change toward the myopic side (increase in refraction) begins with the seventh year and continues at a high rate until the age of 14, slows down through 15 and 16 and slows still more during the ages 17, 18 and 19. In the last period it is little more than half the rate found between the ages of 7 and 14. After 20 there is practically no change. For ages beyond 31 my material is too small to permit reliable deductions.

The curve found suggests that changes in refraction after the age of 7 depend on factors which affect growth. This implication supports

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Refraction Changes in the Eyes of Children Under Six Years of Age, Arch. Ophth. **7**:294 (Feb.) 1932; Homatropine and Atropine Cycloplegia, ibid. **7**:389 (March) 1932. Brown, E. V. L.: Apparent Increase of Hyperopia up to the Age of Nine Years, Am. J. Ophth. **19**:1106 (Dec.) 1936; Net Average Yearly Changes in Refraction of Atropinized Eyes from Birth to Beyond Middle Life, Arch. Ophth. **19**:719 (May) 1938. Marron, J.: Cycloplegia and Mydriasis by Use of Atropine, Scopolamine and Homatropine-Paredrine, ibid. **23**:340 (Feb.) 1940.

the biologic theory rather than the use-abuse theory. If the latter were correct the trend toward myopia would increase markedly. The net average decrease in refraction (increase of hyperopia) up to the end of the sixth year is also in definite disagreement with the view usually held that human beings are born far sighted (hyperopic) but that the hyperopia at once begins to decrease. I have no explanation to offer for the finding.

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COLOR DISCRIMINATION IN INDUSTRY

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The importance of color vision in many industrial jobs has long been recognized. A logical job analysis, even without experimental evidence, is sufficient to convince almost every one that color vision is essential in such jobs as matching samples of various colored materials and operating mobile equipment for which the operating signals are colored lights. It is not our purpose in reporting the present experimental work to reemphasize the importance of color vision among employees on jobs of this type. Our purpose, rather, is to summarize certain data, based on the results of a survey of approximately 7,000 industrial employees, which show that (*a*) color vision apparently is important for certain industrial jobs that have not been identified in logical job analyses as requiring color vision, (*b*) color vision is markedly reduced with increasing age and (*c*) color vision exists in greater or lesser amount on a continuous scale from color blindness, at one extreme, to keen color sensitivity, at the other. The evidence leading to these three conclusions, and the industrial implications of this evidence, will be considered in turn.

The visual survey was conducted in a sheet and tin mill under the medical supervision of one of us (H. S. K.). Several tests were included in the battery, and among these was a simple four item red-green color discrimination test. Later analysis of the results revealed that the tests (including the one of color vision) could be improved in a variety of ways. Indeed, one of the major purposes in conducting the survey was to determine from first hand extensive experience just what types of visual tests would be most suitable for use by laymen in survey and employment office testing. Specific weaknesses of the test of the color vision that came to light will be discussed later. For the present, it need only be mentioned that the most difficult item in this test was passed by 55.3 per cent of the 7,000 employees tested. One should not infer that the remaining 44.7 per cent of the employees, who failed this test item, were color blind; but it can be concluded that they had less ability to discriminate colors than the 55.3 per cent who passed the test.

Read before the Section on Ophthalmology at the Ninety-Third Annual Session of the American Medical Association, Atlantic City, N. J., June 11, 1942.

It should be kept in mind that the present results are based primarily on the test of red-green discrimination. An additional four item test of yellow-blue discrimination was given to 500 employees, but this number was not considered sufficient to justify a detailed statistical analysis of yellow-blue test results. It was interesting to note, however, that among the 500 employees who were given both tests were found 22, or 4.4 per cent, who missed no items on the red-green test but did miss the most difficult item on the yellow-blue test. In other words, 4.4 per cent of these 500 employees were more deficient in yellow-blue discrimination than in red-green discrimination. This finding suggests that, contrary to general belief, weakness in yellow-blue discrimination may, at least in some cases, be unique in that it may be present without a significant weakness in red-green discrimination.

Returning to the results of the red-green test as a first means of determining for all employees in the mill whether deficient color vision is in any way associated with poor job performance, all employees were separated into three groups according to their merit ratings on job performance which had previously been made by supervisors. The three merit rating groups were high, average and low. The high group contained the third of the employees who had been rated as best in performance on the job; the average group contained the middle third according to the merit ratings, and the low group contained the bottom third according to these ratings. The employees were further subdivided into five-year age groups. This double division identified for each five-year age group three groups of employees according to their rated general ability on the job.

The percentage of employees among the highest and the lowest merit rating group at each age level who passed the most difficult item in the test of color vision is graphed in chart 1. Except for the two extreme age groups (neither of which contained many employees) it will be noted that at every age level the group of employees who had been rated high in job performance contained a definitely larger percentage of employees who passed the test of color vision. The curves in chart 1 are based on 4,000 men employees, 2,000 in the high-rated group and 2,000 in the low-rated group. The curve for employees in the middle third according to the merit rating is not shown, because it adds nothing to the general picture.

These results show that a fairly high degree of color vision is much more frequent among general mill workers who have been rated high in job performance than among employees who have been rated low, in spite of the fact that not over 5 per cent of the jobs involved were considered to require color vision.

At least two possible explanations of these findings may be offered. It is possible that color vision is required of many more employees than any one has recognized. Every employee must recognize certain danger signals, whether he is operating mobile equipment or not; every employee must be able to distinguish between company forms and charts, which are often printed on different-colored paper for ease in identification, and every one in the mill should perhaps know something of the color code which identifies the plumbing in this plant. It is possible that inability properly to handle these related aspects of the job accounts for the lower merit rating given to the employees with deficient color vision.

A second possible explanation may be that color vision is a barometer of general health and physical condition to such an extent that employees with reduced color vision are also suffering from general physical debilitation to such an extent that they are in general unable to perform their

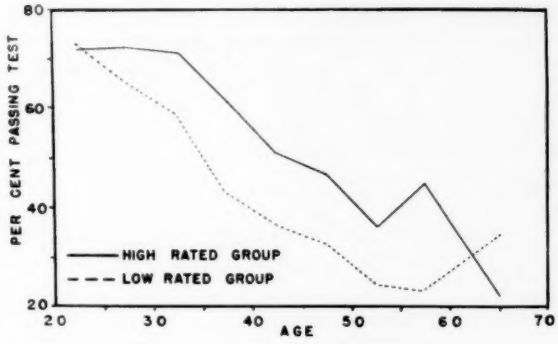

Chart 1.—Differences in red-green color discrimination between employees with high and employees with low job performance ratings.

respective jobs satisfactorily and hence receive low ratings from their supervisors as well as poor scores on the test of color vision. Dr. T. D. Allen, of Chicago, has called our attention to the fact that excessive use of tobacco or alcohol often results in a definitely reduced sensitivity to color. The employees failing the test of color vision and also receiving the low merit ratings may be the dissipaters, and their dissipation may account for both their deficiencies.

Whatever may be the final explanation, the statistical conclusion is unescapable that the least desirable employees from the point of view of general job performance are the ones who failed the color vision test, and it follows as a natural corollary that such undesirable employees can be identified by the color vision test before they are put on the job. Industry has in such a test a reasonably satisfactory device for predicting whether a man hired for general mill work will be successful on the job.

Another approach to this general subject, with particular reference to a few specific jobs, was made by determining the percentage of employees on each of seven specific jobs who were able to pass the test of color vision. These results are shown in the accompanying table.

Two of the percentages, the one for foremen and the one for clerks, show a statistically significant difference from the plant average, i. e., a difference that cannot be explained in terms of random or choice variations and must therefore be considered as indicating a real difference between the figure for all employees and that for employees on the job in question. Employees on both those jobs passed the test of color vision more frequently than employees selected at random from the mill. It thus seems that something about those jobs calls for color vision, or some characteristic associated with color vision, more frequently than most other jobs call for this ability. The findings cannot be explained by

Percentage of Employees on Different Jobs Who Passed the Color Vision Test

	Number of Employees	Percentage Passing Test
All employees in the plant	7,000	55.3
1. Foremen	320	66.5 *
2. Clerks	590	79.2 *
3. Electricians	430	59.6
4. Machinists	380	60.0
5. Cranemen	250	57.0
6. Hookers	250	59.1
7. Laborers	710	52.7

* Differs significantly from the plant average.

differences in age of employees on different jobs, because differences in age were eliminated statistically.

As in the case of the relationship between color vision and merit rating for all mill employees, it is one thing to find the fact and something else to explain it. It is not too difficult to understand why the foremen group contains a large percentage of men with good color vision. These men must be familiar with all kinds of details pertaining to their jobs, and among these details are the color code of the plumbing system, colored lights and signals and the colored charts and forms. It is more difficult to explain why clerks are so superior in this respect, particularly in view of the fact that they not only are superior to the average mill employees but are definitely superior to the foremen, who, as already mentioned, are superior to the average mill employees.

But findings based on samples of employees as large as those here reported are likely to stand even though we are unable at the present time to explain them. It is clear that some selective factor is operating to prevent persons with deficient color vision from holding jobs as foremen

or clerks. Here, then, are two specific jobs with a requirement of a high degree of color vision that is not revealed in an ordinary job analysis.

A second finding apparent in chart 1 is that with increasing age a successively smaller percentage of employees is able to pass the test of color vision. This is shown both for the group rated high and for the group rated low in job performance. It is shown more simply for all employees in the mill in chart 2. Seventy-four per cent of employees between the ages of 20 and 25 passed the test of color vision. Only 32 per cent of employees above the age of 55 passed the test. This is a ratio of more than 2:1 for employees at the extremes in age who had the degree of color vision necessary to pass this test.

Color vision, apparently, is not like a foot or an ear, something which, barring accident, if one has at age 20 one will continue to have at age 70. Rather it seems to be more similar to near point accommodation. One

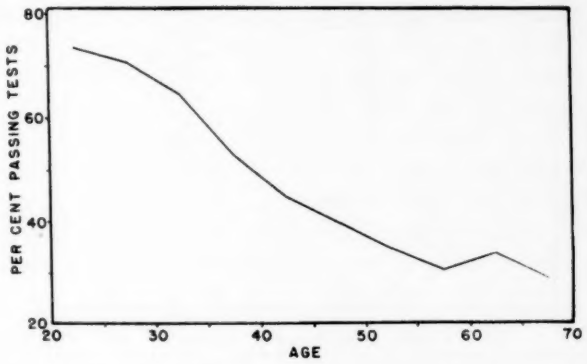

Chart 2.—Changes in red-green color discrimination with age. The patients tested numbered 7,141.

loses it as one grows older, and the less one has at an early age, the more quickly the loss with age will reduce one's functional efficiency on any job that requires this particular skill. There are many industrial jobs for which this loss can be serious indeed. Consider, for example, the operation of a modern color-printing press. The mechanical and operative aspects of this job require, on an average, ten years for complete mastery. If one begins one's training or apprenticeship at the age of 20, one's most productive years will ordinarily occur after the age of 30. But if the color vision deteriorates during this period to an extent that makes one unable to handle the color aspects of the job, one's mechanical and operative training for ten years is an irreparable loss both to one's company and to oneself.

Many other industrial jobs require both long experience and color vision. The case of the foremen cited is a typical example. The facts

concerning the loss of color vision with age should caution every personnel manager to be sure that every employee who receives special training should have enough excess color vision at the outset so that even after some of this ability has been lost with age, enough will remain for satisfactory job performance.

A final conclusion indicated by our present results is that color vision may be present in greater or lesser amount. It is not an "all or none" proposition. Persons are not necessarily normal in color vision or color blind. Indeed, the great majority of persons are somewhere between the two extremes.

This conclusion is suggested by the successively decreasing percentage of persons able to pass the color vision test at different age levels. It is proved by the data graphed in chart 3. This chart shows the percentage of employees who were able to pass one, two, three and four items of a

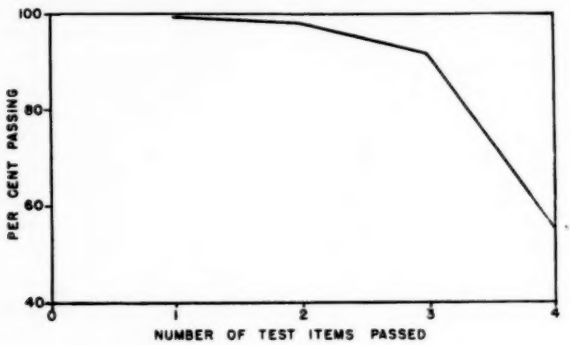

Chart 3.—Percentage of employees passing test of red-green color discrimination at different levels of difficulty.

four item color test. Ninety-nine per cent of the employees passed the first item. This means that practically every employee had some color vision. Ninety-eight per cent passed two items, showing a slightly greater degree of color vision; 90 per cent passed three items, evidencing a still higher degree of color vision, and 55 per cent passed all four items, showing a still higher level. It is doubtful whether an arbitrary point should be set on the continuum and accepted as the dividing line between the color blind and the normal. No employee tested was completely color blind. Indeed, Fry [1] reported in 1930 the eighth case of total color blindness that has appeared in the American literature and one of about 125 such cases that have been reported in all literature throughout the world. Total color blindness is very rare indeed. But probably just

1. Fry, W. E.: Congenital Total Color-Blindness, Am. J. Ophth. **13**:1064-1065, 1930.

as rare is the extremely high degree of color sensitivity possessed by a few gifted painters or other persons whose work is vitally dependent on a high degree of this capacity.

The implications of our results for industry are that what is needed is a test that will measure the amount of color vision possessed by an applicant or an employee and not just classify him as color blind (if he is in the lowest 4 to 8 per cent of the population) or normal (if he is in the upper 92 to 96 per cent). It is not enough for an industry to know that a man being trained as a color pressman is in the top 96 per cent. Among persons in this group will be a few, those near the bottom, whose color vision will be inadequate within two or three years. To measure color vision on such a scale is equivalent to measuring weight in two categories, every one below 120 pounds being called light and every one above this point heavy. The present research has resulted, among other things, in a statement of the specifications for a newly conceived and graded test of color vision that measures the amount of color vision all the way up the scale. This test is being checked and rechecked, tried out with industrial employees, and when made available commercially should be of considerable help in aiding both industry and the medical profession in detecting changes in color vision that are of vital importance for job success or that may occur as a result of disease or therapeutic treatment.

In summary, tests of color vision administered to approximately 7,000 industrial employees have shown that (1) color vision is often necessary for employees in jobs which have not been known to require color vision, (2) color vision shows a marked reduction with increasing age, making it necessary for an industry to be doubly sure that persons being trained for certain jobs have an excess of the ability when the training is begun and (3) color vision exists in greater or lesser degree and is not an "all or none" proposition of the type assumed by the classification of persons into a color blind or a normal group.

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ABSTRACT OF DISCUSSION

Dr. Alfred Cowan, Middlesboro, Ky.: This investigation by Dr. Tiffin and Dr. Kuhn has certainly brought out some surprising results. My first reaction is an intense desire to learn the method of examination, based primarily on red-green discrimination, by which such fine determinations can be made. It is well known that what is generally spoken of as partial color blindness exists in varying degrees, probably similar to the varying degrees of visual acuity, and probably if there were some means of measurement color vision could be noted as visual acuity is noted. But as yet there is no clinical method of testing color vision which is comparable to the tests for visual acuity.

By the unrefined office methods ordinarily used for testing color vision on the basis of red-green-blue discrimination, the best that can

be stated is that a person is a monochromat, a dichromat, an anomalous trichromat or a normal trichromat. It will be extremely interesting to learn of a reliable test of the color vision, based on the Young-Helmholtz theory, consisting of four items of which the most difficult would screen out 44.7 per cent. I am sure most ophthalmologists will agree with me that according to the current conception this is an extremely high percentage of color defectives. It seems, too, that if the same results would hold generally, relatively few persons would qualify as examiners. Also, while it is known that color discrimination is reduced because of certain senile changes which take place with increasing age, it seems remarkable that the authors have found that at the age of 45 the percentage of failures has already risen to about 57 per cent, as shown in chart 2. These figures are somewhat upsetting in view of the fact that almost without exception master dyers are well past middle age.

Of greatest importance, however, is the conclusion of the essayists, after analyzing their data, that they have devised a practical means of estimating the aptitude, efficiency and intelligence of workers in every type of industrial job by the application of their four item test of the color sense.

DR. PETER C. KRONFELD, Chicago: This work by Dr. Tiffin and Dr. Kuhn is primarily intended for and is of special interest to those ophthalmologists who act as advisers in matters of the visual requirements for employment in industry.

The study has brought to light at least two findings of general ophthalmologic interest, namely (1) a higher incidence of anomalies of color sense than has been reported in other populations and (2) a dependence of this incidence on the age of the employees.

In order to be able to evaluate these findings, one has to know a little more about the test which in the paper is described as "a simple four item red-green discrimination test."

The test was conducted with cards put out by the Keystone Company and apparently designed on the same principle as the pseudoisochromatic plates of Stilling or Ishihara. To me the four items seem very easy, and I do not understand why almost one half of the entire group, or one fourth of the persons under 30 years of age, failed to pass the test. Peculiar lighting may have made the test more difficult. It would seem significant that if the most difficult item was omitted from the test the incidence of anomalies of the color sense would be reduced to roughly 10 per cent, a figure which is in close agreement with the results obtained with other methods in other populations.

The apparent incidence of deficiencies in the color sense depends to a large extent on how fine a test is used. Theoretically, at least, it would be possible to design a test which would be passed by a still smaller percentage of persons than that reported here.

The fundamental question, therefore, is, What degree of color discrimination is necessary for efficient performance of a certain job? If the answer to that question were available, it would be fairly easy to devise a test of adequate fineness and accuracy. For quantitative studies of the color sense of the accuracy required in the color printing industry, it would seem to me that the most accurate and dependable test should

be used, and in my opinion this should be a test based on equations between mixtures of spectral red and green on the one side and spectral yellow on the other side.

The authors have stressed the quantitative nature of the ability as well as the inability to discriminate colors on a continuous scale. In principle this is true, since with accurate methods the occurrence of many gradations or transitional forms between the normal trichromatic, the anomalous trichromatic, the typical dichromatic and the monochromatic color systems have been demonstrated.

With regard to the apparent diminution in the ability to discriminate colors which occurs with increasing age, I wonder if the authors have any explanation to offer for this phenomenon. Since it occurred in eyes free of any gross disease, I would be inclined to correlate it with the absorption of light of short wavelength by the crystalline lens, which occurs in the eyes of older persons to a greater degree than in those of young persons.

Whether or not a prospective employee in a color-printing plant is likely to acquire a marked degree of this absorptive form of color deficiency in later years would seem to me difficult to determine at the age of 20, but that is the time when it should be decided whether the applicant is a good or a bad risk from the viewpoint of color discrimination. The degree of color sense which the applicant possesses at the age of 20 would, to me at least, not be indicative of or related to the degree of color deficiency that might result from changes in light absorption by the lens later on. Besides, such changes in the lens occur so slowly that an experienced employee could be expected to make the necessary adjustments, consciously or unconsciously.

DR. JOSEPH TIFFIN, Lafayette, Ind.: I should be the first to agree, and I am sure Dr. Kuhn would also agree, that we are not satisfied with this particular testing device for segregation of industrial employees, whereas we would both agree that a test which permits only 55 per cent of a large group of persons selected at random to pass it is, in general, a fairly stiff test or a fairly fine test.

We feel rather strongly that, fine as the test is, it is still not fine enough, because there are a good many types of industrial jobs (jobs calling for a high degree of color discrimination) in which one wants to know not that a person is in the best 55 per cent of the population but whether or not he is in the best 10 per cent or the best 5 per cent. It would be exactly the same as if one had a thermometer that only measured the temperature reached on 45 per cent of the days. It would be a fine thermometer for 45 per cent of the days, but on the other 55 per cent it would leave quite a bit to be guessed.

Industrial classification of employees clearly indicates that there are many cases in which one wants to measure accurately the exact degrees of color vision that the upper 55 per cent have. That is exactly what we have striven toward in the construction of a new test—which I might say by way of anticipation does definitely accomplish the desired result.

ANCIENT MEDICAL JURISPRUDENCE WITH SPECIAL REFERENCE TO THE EYE

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In all ancient systems of law, a sense of justice demanded the infliction of the same pain and the same loss on the aggressor as he had inflicted on his victim. Hence the prominence of the principle of *lex talionis,* or "measure for measure," in ancient law. The Old Testament legislation "an eye for an eye" is not the first conception of ancient justice. About twelve hundred and fifty years prior to the Mosaic code, Hammurabi (reigned 2285 to 2242 B. C.), king of Babylonia, enacted laws based on the principle of "an eye for an eye." [1] His interpretation of *lex talionis* in many instances was more rigid and unrelenting than the Mosaic conception of retaliation. According to the Babylonian code, for example, when a man struck a pregnant woman (free born) so as to cause the death of the fetus by miscarriage, the daughter of the assailant had to be executed [2]; or when a builder contracted to build a house and the house collapsed and killed the son of the proprietor, the builder's son had to be put to death.[3] In these cases the concept of redress goes far beyond the penalty of the Mosaic law. According to the Bible, such an interpretation of the concept of retaliation is impossible, for the Bible distinctly states: "The fathers shall not be put to death for the children, neither shall the children be put to death for the fathers; every man shall be put to death for his own sin." [4]

The severity of the ancient Babylonian code was applied equally to the surgeon and to the oculist, whose practice was regulated by law. Thus, "If an *asum* (physician) opens a *negabati* of the eye of a freeman and causes the loss of his eye, the physician's hand shall be cut off; if he has caused the loss of an eye of a poor man or slave, he shall pay 1 *mina* of silver." [5] It is evident that the penalty for malpractice in the case of a freeman was not "an eye for an eye" but a hand for an eye. The construction of the principle of *lex talionis* in the case of the oculist applied to the hand that caused the injury even if the mishap

1. Exodus 21: 24.

2. (*a*) The Oldest Code of Laws in the World, translated by C. H. W. Johns, Edinburgh, T. & T. Clark, 1903, pp. 45, nos. 209-210. (*b*) Exodus 21: 22-23.

3. Footnote 2 *a*, p. 49, no. 230.

4. Deuteronomy 24: 16.

5. Footnote 2 *a*, p. 46, no. 218.

was the first of that surgeon. This harsh and unrelenting retaliation, if one may read between the lines, was to prevent the oculist from causing injury to another patient. The Persians, who conquered Babylonia, were more lenient to the surgeon; they required three unsuccessful operations before a practitioner was even disqualified from the practice of surgery; there was no question of physical retaliation.

Incidentally, there is a difference of opinion among Assyriologists as to the meaning of the term *negabati,* for the treatment of which the Babylonian *asum* was physically responsible. Johns stated the belief that a *negabati* was an abscess (possibly of the lid or of the lacrimal canal). Magnus expressed the opinion that it was a cataract, because an abscess of the conjunctiva or of the canal would hardly cause blindness. Von Oefele, who agreed with Magnus, rendered the paragraph of text 218 of the code of Hammurabi as follows: "If an *asum* opens a star (cataract) of a free-man with a pointed knife of copper and causes the loss of a free-man's eye, one shall cut off his hand." [6] It should be pointed out that the fact that a knife was mentioned in connection with the cataract operation indicates that the Babylonian oculist favored paracentesis rather than *reclinatio lentis* (the couching, or expressing, operation, practiced among ancient oculists). Because of the dislocation of the cataractous lens into the vitreous, ancient oculists seldom saw a cataractous lens, and they came to the conclusion that the impairment of sight was produced by injurious humors of the brain flowing through the hypothetically hollow optic nerve into the eyeball, where they settled between the iris and the lens. The density of the latter observed in dead human beings and animals was believed to be a postmortem change.

In one of the earliest records of Egyptian ophthalmology, known as the Papyrus Ebers, cataract was described as "mounting of water in the eye." Hippocrates and even Galen called it by a similar name; hence the word cataract ("a great waterfall"). The Romans knew it by the term *suffusio oculorum.* Celsus, who described cataract under that name, prescribed as its treatment *reclinatio lentis* (couching). This treatment of cataract appears to have been the prevailing procedure among the Hebrews of Palestine and Babylon. There were professionals who occupied themselves with couching of cataract (*icutto*). These specialists evidently had a lucrative practice, for cataract has always been the source of a large proportion of blindness in the Middle East. Said Rabbi Levi, "Unhappy is the land whose *icutto* (cataract couchers) themselves have one eye" [7] (conveying the idea that if the surgeon had one eye blinded by cataract he was apt to injure rather than benefit the patient).

6. Footnote 2 *a*, no. 250.

7. Ikutta Leviticus Rabba, chap. 5.

Even the barber (*gallebu*) had a legal status in the Babylonian code. In addition to his tonsorial manipulations on the black beards of the Babylonian nobles, he practiced venesection, cupping and leeching, marked slaves on their arms or foreheads to identify them and probably performed the operations of circumcision and dental extraction.

The legal status of the barber was circumscribed in Babylonia, so that he could not overstep the functions assigned to him. In this respect, medieval Europe, where the barber usurped the role of the surgeon, had much to learn from the Babylonians of four milleniums back. Medieval Europe might also have taken a hint from the Babylonians with reference to theurgic medicine. The code of Hammurabi recognized only the surgeon, not the physician, for the practice of internal medicine was in the hands of the priests and magis and the code dealt only with concrete matters and not with religious or spiritual problems.

Like all other nations of antiquity, Babylonia could well understand illnesses as caused by violence but could not conceive of them as normal incidents of life. The Babylonian believed them to be produced by the intervention of extraneous metaphysical forces, such as demons and ghosts of the dead, and of course the physician could not be held responsible for these offenders. When it came to surgery, the one who performed the operation was responsible for the damage done. The patient was even allowed to claim the return of the physician's fee if his operation was not successful. There was no difference between the status of the surgeon and that of the builder of a house, who was held responsible if the building collapsed. (The code of Hammurabi, previously quoted, emphasized that the fee was to be paid on cure of the eye; it may be assumed that when there was no cure there was no pay.) It is questionable whether these rigid laws applied to military surgeons, who must have occupied a prominent position in old Babylonia in view of the incessant wars and continuous military campaigns of the Babylonian kings with their neighboring nations. Archeologists and historians identify Hammurabi with Amraphel, King of Shinar (Genesis 14:1); if this is correct, he was the head of four allied nations who carried on war against five axis nations.

About fifteen hundred years later, during the reign of Sargon, or Saru Kinus ("the legitimate king"), the surgeon played an important role. He was directly under royal command. A surgeon named Ardana in a letter to the king wrote:[8]

> Concerning the patient with the bleeding from the nose, the Rabmugi told me that bleeding occurred yesterday toward evening. The bandage of the patient is a surgical mistake. For it is attached to the nostrils, so that it hinders respiration,

8. Leix, A.: The Medical Knowledge of Babylonia, Ciba Symposia **2**:674 (Dec.) 1940.

while the bleeding can still go on through the mouth backwards. Let the nose be tamponaded, so that the passage of air through it will be prevented and the bleeding stopped.

From this letter it may be inferred that otolaryngology was already developed in the seventh century B. C. Even medical knowledge, which was previously considered a priestly craft, was adequate at this period. Fredrick Kuchler has deciphered and published a medical compendium found in the library of Ashurbanipal, which gives an interesting insight into the knowledge of medicine possessed by the Assyrians. This text was evidently compiled from different sources. The Assyro-Babylonian pharmacopeia, according to Von Oefele, contained 250 medical plants, 120 mineral substances and 180 other drugs.[9]

Herodotus stated that medical practice in Egypt was legally regulated.[10] According to the Hermetic books of Thot, if a patient's death resulted from any deviation from the set rules prescribed for the physician, the latter was guilty of a capital offense. Aristotle a century later observed that physicians in Egypt were allowed to alter the prescribed method of treatment after the fourth day of sickness if the patient did not improve. The risk of his medicine's proving injurious was then the physician's own risk.[11]

In China, if death occurred during medical treatment, the doctor was held responsible. If death followed an operation, it was considered a murderous act, and the old law of *lex talionis* was enforced; the physician had to pay with his life. The death penalty, however, could be ransomed for money if this was satisfactory to the relatives of the dead. A Chinese physician who lost a royal patient in the course of treatment was buried alive with his dead patient. This practice was discontinued in the year 1650. The failure of a physician to save the life of a member of the Chinese royalty, however, was punished with death until comparatively recent years, regardless of blame.

Chinese emperors and head ministers of the government engaged in the practice of medicine as an avocation or hobby. Royal physicians as a rule did not charge fees. Grateful patients, however, donated gifts to the state for their generous actions. A considerable amount of healing is still carried on in China by the elders of a clan (i. e., grandfathers and grandmothers), who use home remedies that have been accumulated through the ages.

The Mosaic code prescribes "one manner of law" [12] and does not attempt to give a separate legal status to the *rophe* (physician). It is

9. Leix,[8] p. 679.

10. Herodotus: Euterpe, translated by H. Cary, New York, American Book Company, 1847, vol. 2, p. 125.

11. Aristotle: Political Theories of Aristotle with an introduction by W. L. Newman, London, Oxford University Press, 1910, vol. 3, p. 15.

12. Leviticus 24: 22.

questionable whether the law "an eye for an eye" was ever taken literally by the ancient Hebrews. The only injunction against ransom is in the case of a capital offense: "Ye shall not take ransom for the soul of the murdered." [13] There is no injunction against payment of money for injuries sustained. Even the Sadducees, who customarily adhered to the letter of the law, appear to have disregarded the literal application of "an eye for an eye." In their disputes with the Pharisees, referred to in the Mishnah,[14] no allusion is made to any differences between the two parties regarding the form of retaliation for bodily injuries.

The case of the surgeon who has intentionally injured his patient is embraced in the law of assault and battery of Exodus: "He shall pay for the loss of his time and shall cause him to be thoroughly healed." [15] The *lex talionis* in the Old Testament was applied only in the case of murder. Then the aggressor had to pay life for life; there was no ransom for the murderer. It should be recalled that among many nations of antiquity and among Germanic tribes as late as the Middle Ages, atoning was allowed even for murder. *Vergild* (ransom) was paid to the heirs of the slain man. On this point, the Mosaic law is emphatic: "Ye shall take no ransom for the life of a manslayer."

Furthermore, the Old Testament law is against mutilation as a form of retaliation. There is only one instance to the contrary, the meting out of punishment to a woman who commits an impropriety when she takes the part of her husband during a fight with another man. The Bible specifies that such a woman's hands are to be cut off.[16] This punishment cannot be classed under the law of *lex talionis*. It harks back to the Nuzi law dating from the fifteenth century B. C.[17]

According to Josephus, the principle of "an eye for an eye" was never enforced by ancient Hebrews when the assault did not end in loss of life. "Although," stated Josephus, "maiming is avenged by the talio, the sufferer may receive compensation, and is allowed by law to estimate the amount." In postbiblical times, petty assaults were generally dealt with by fines, these varying according to the dignity of the injured person—a class distinction that is reminiscent of the Babylonian code.[18]

The Talmudic jurists unanimously agree that the Biblical law "an eye for an eye" is to be interpreted in a figurative sense and that the

13. Numbers 35:31.

14. Mishnah Yadayim 4:6.

15. Exodus 21:19.

16. Deuteronomy 25:11-12.

17. Gordon, C.: A New Akkadian Parallel of Deuteronomy 25:11, 12, J. Palestine Orient. Soc. **15**:29-34, 1935.

18. Josephus, F.: The Jewish Antiquities, translated into English by W. Whiston, Boston, S. Walker, 1833, book 4, chaps. 8 and 35.

loss of an eye may be ransomed for money. They are of the opinion that retaliation in the Bible applies only to one who commits murder and that all lesser crimes can be expiated by financial recompense. The Talmudists argue that the book of Leviticus distinctly states "Ye shall have one manner of law" [19] and that in the case of "an eye for an eye" this injunction may often be impossible to carry out, as when a blind man puts out the eye of a man possessing sight.

The laws pertaining to ophthalmic practice are fully discussed in Talmudic literature. Thus, if a patient says to the physician, "Blind my eye because it is very painful" or "Cut off my hand because it is painful" and the physician follows the directions of his patient, he is free from liability.[20] (It is assumed that the physician has examined the eye of the patient and found it necessary to enucleate the eye.) This freedom from liability is also granted to the *rophe* even when he does not obtain permission from the patient to operate on his eye as long as it can be proved that he has the interest of the patient's health in mind. But if the patient is not sick and the physician at his request blinds him or cuts off his hands, then the physician is held responsible.[21]

When one says to the physician, "Blind the eyes of X, I will be responsible for damages," then the physician is responsible, because no one can legally become an agent for doing evil to another.[22] The one who engages the physician is equally responsible because he caused the spiritually blind (the physician) to stumble.[23] If a slave says to his master (who is a physician), "Put medicine in my eye" and the medicine blinds him, the master is held responsible and must set him free.[24] If the master injures the eye of a fetus (during delivery), he is not liable.[25]

If the patient dies in the course of treatment, even though there has been no negligence, the physician has to depart to the "free cities" and remain there as long as the high priest is alive.[26] These laws applied to physicians who were authorized and licensed by the *beth din* (board of judges) [27] but not to those who practiced without a license. The latter were considered as laymen and were punished as such.

The Rabbinic legislation with respect to physicians is summed up as follows: "A doctor, authorized by the *beth din* to practice medicine,

19. Leviticus 24:22.
20. Mishna: Baba Kama 8:7; Talmud: Sanhedrin 84 *b*.
21. Mishna: Baba Kama 8:7. Talmud: Baba Kama 93 *a*.
22. Talmud: Baba Kama 8:7.
23. Yad Hazakah Hilchoth Chobel 5:13.
24. Talmud: Kidushin 24 *b*.
25. Talmud: Kidushin 24 *b*.
26. Exodus 21:14. Tosefta: Makkoth 2:5.
27. Tosefta: Baba Kama 6:17.

who unintentionally injures his patient in the course of treatment, goes free."

On the other hand, if the injuries are intentional, the physician is responsible. If it is a question of misjudgment, the physician is not liable because of *Tikon Haolam* (i. e., if a doctor is held responsible for misjudgment, no one will take up the practice of medicine as a profession).[28]

When it is difficult to decide between a legitimate mistake and negligence, the physician gets the benefit of the doubt, for "he surely will be punished in the world to come if he is at fault."[29] If it can be proved that a physician wounded a patient more than necessary during an operation, then he is responsible.[30] "If the physician injures his patient intentionally, he is of course responsible to pay for damages." These, according to the Jewish law, are fivefold and consist of *nezek* (remuneration for loss of function), *tzaar* (for pain), *ripu* (for medical services), *shebeth* (for loss of time) and *bosheth* (for shame or disfigurement). Payment of damages depended on the verdict of the judges. The liability for malpractice is nullified if the patient does not carry out the orders of the physician.[23]

Practitioners of venesection employed by the community were discharged even if their contracts had not expired if they neglected to treat their patients kindly. Rabbi Papa applied the same regulation to practitioners of circumcision who were employed by the community.[31]

In contrast to the healer in ancient Babylonia and China, against whom there were rigid laws, the Hindu physician enjoyed privileges and kind consideration from the public at large. During the early Brahmanic period (800 B. C. to 100 A. D.), when medicine was a part of priestcraft, there was no question of punishment for malpractice. Both disease and its treatment emanated from the gods, and the priests, who only carried out the wishes of the deity, could certainly not be penalized. After the metaphysical concept with reference to medicine was discarded, the Hindu physician did not need a legal code to protect him from punishment in the case of injury inflicted on his patient during his medical or surgical practice. The medical regulation to which the physician had to submit was sufficient protection against injustice done to himself or to his patient. The Hindus adopted a code of medical ethics noted for its sagacity and earnestness. The ethical and professional requirements demanded from the medical practitioner were of such a nature that if they were followed medical and surgical mishaps

28. Tosefta: Gittin 3:6.
29. Tosefta: Baba Kama 6:17.
30. Tosefta: Baba Kama 9:11.
31. Talmud: Shabbath 133 *b*.

would be reduced to a minimum. Some of the Hindu's ethical doctrines, for example, were:

A physician, who desires success in his practice, his profit, a good name and finally a place in heaven, must pray daily for all living creatures.

Let his speech be short, clear, pleasant; his transaction in the house should not be bruited abroad.

The latter advice is also found in the Hippocratic oath.

Surgical procedures attained a high position among the Hindus. Operations were performed at the beginning of the warm season. The skin was rubbed with a pledget of cotton saturated with a suitable medicament.

Dressing of wounds was done in a most sanitary manner. The wounded on the battlefield were quickly picked up and carried into a tent, the bleeding stayed and the wound washed with an anodyne oil. Cautery was frequently used: "The fire cures disease which the knife, physic and herbs do not cure."

The Hindu mode of extracting cataract has survived to the present day. Careful attention was paid to the patient's age, mode of life, habits, pulse, body temperature, color of the skin, condition of the eyes, urine and feces, strength of the voice and respiratory sounds and especially to his intelligence. The last, according to Susruta, was important: "Stupid people are more easily cured than intelligent individuals because the first are more obedient to the instructions of the physician." [32] If the disease was of an incurable nature, the Hindu physician refused treatment but advised the patient to prepare for the occasion when his soul would be united with God. The physician was advised "to wear his hair short, to keep his nails clean and cut close, and to wear a sweet-smelling dress." The superior ethical and practical regulations of the Hindu physician undoubtedly commanded the respect and admiration of his patients and reduced malpractice proceedings to a minimum.

Garrison [33] stated:

Whether the Hindus influenced Greek medicine before the time of Alexander the Great or were themselves influenced by it is not known; but it is certain that, at the time of Alexander's Indian expedition (327 B. C.), their physicians and surgeons enjoyed a well-deserved reputation for superior knowledge and skill. Some writers even maintain that Aristotle, who lived about this time, got many of his ideas from the East.

Be that as it may there is a great similarity in the medical concepts of these two ancient peoples. Both separated metaphysics from medicine at an early date and both developed a superior code of ethics.

32. Allen, T. H.: Science History of the Universe, Medicine, New York, The Current Literature Publishing Company, 1909, p. 115.

33. Garrison, L. H.: An Introduction to the History of Medicine, ed. 2, Philadelphia, W. B. Saunders Company, 1917. p. 61.

In Greece before the sixth century B. C., as in India, medicine was practiced by priests in temples. When the cure failed, the patient did not lose faith in either the powers of the gods or the skill of the Asclepiads. After the dawn of Greek philosophy, priestly medicine commenced to decline, at least among the cultured Athenians, who began to investigate the causes of natural phenomena and particularly those of disease. Various theories were advanced by medical philosophers of that period. Empedocles introduced the doctrine of the four elements, earth, air, fire and water, as the basis of all things. This theory finally led to the humeral theory of disease first elaborated by Alcameon (the discoverer of the optic nerve) and the school of dogmatists. The dogmatists were of the opinion that life depended on the proper proportion and action of the four elements in the body. On the other hand, Democritus theorized that atoms filling the air entered through the pores of the body and were the deciding factors of health and disease. This philosophy was accepted by the medical school of methodism. Pythagoras defended the concept of demonology as the basis of abnormal bodily conditions. These thinkers, having deprived the gods of being the primary cause of all bodily ailments, separated medicine from religion and elevated medicine to the status of an art and the physician to the standing of a creative artist. In this capacity the physician could not longer claim immunity for errors exercised in the course of his professional work.

The Pythagorean society, founded by Pythagoras, did much to regulate the practices of its members, but many of them ultimately became greedy for gain, and the members of the society during their founder's own lifetime were frequently subjected to persecution and dispersed by angry mobs who were looking for retaliation against improper medical attention.

Hippocrates in his law complained that "in the cities there is no punishment connected with the practice of medicine except disgrace, and that does not hurt those who are familiar with it." He further remarked: "Medicine is a noble art, but owing to the ignorance of those who practice it and those who inconsiderately form a judgment of them, it is at present far behind other arts."

The regulations following the advance of medicine in Greece by Hippocrates were rather of an ethical nature and were not enforced by state ordinance. In the Hippocratic oath, the duties of a physician to his patients are made paramount; all else is of minor importance. In his maxims, the ethical duties of the physician are still further elaborated: "The physician should benefit or at least not injure." "Where there is love for the arts there is also toward man." "The physician's duty is to watch and foresee the change in his patient, to assist and not to hinder nature, so that the sick man may conquer the

disease." The deep sense of duty engendered in the medical practitioner tended to protect both the patient and the physician.

Philament, a Greek writer, well expressed the Greek attitude toward the physician in his time when he said, "The physician has a right to kill and not to be killed."

Unlike Greek medicine, which occupied an exalted position for eight hundred years beginning with the sixth century B. C., Roman medicine before the Roman conquest of Greece was at a low ebb. The material character of the Romans' daily life drew away their attention from philosophy and medicine to physical culture and bodily enjoyment. According to Pliny the Younger, the Romans got along well without physicians for a period of six hundred years, relying on superstitious religious rites, dietetics and household remedies. The average Roman citizen had for nearly every disease a household god to whom he paid homage in case of sickness. The historian Baas agreed with Pliny that the Romans had no physicians but stated the belief that they had different remedies for all diseases. Their healing art consisted of prayers, magic rites, prescriptions from the Sibylline Books and a large collection of household remedies. Dispensing of home remedies was in the hands of the elders in the family—the grandfathers and grandmothers.

Roman medicine of later days was borrowed from Greece. After the destruction of Corinth (146 B. C.), a large number of Greek prisoners of war, many of whom were physicians, came to Rome. These captives had no particular love for their captors. Marcus Porcius Cato, who was a censor by profession and practiced medicine as a hobby, in a letter to his son warned him against the Greek physicians. Among other things he wrote:

> They have taken a mutual oath to murder all the barbarians with their medicines, but for all that they demand expensive fees in order to make one trust them. In this way they can carry on their sinister game with greater ease. They also class us with the barbarians and thus they sully us in a baser manner than if they had called us old-fashioned. I forbid you to associate at all with doctors.

Almost two centuries later, Pliny complained: "There is unfortunately no law which punishes the doctor if through his fault some one dies. It is permitted to him by our danger to learn for the future, at our death to make experiments, to set at naught the life of a human being." [34]

The antagonism to Greek physicians was so strong that Archagathus, the first celebrated Greek physician to settle in Rome (218 B. C.), was nicknamed *Carnifex* (butcher). The imperial city was the happy

34. Pliny: Historia Naturalis, 29:8; cited by Preuss, J.: Biblisch-talmudische Medizin, Berlin, S. Karger, 1911.

hunting ground for quacks. They went about followed by pupils and patients. The latter were allowed to follow them on the promise that if a cure was effected they would proclaim the skill of their healer. The students went along with these charlatans not so much to receive medical bedside instructions as to impress the family with their learning and skill as teachers of the medical arts. Martial,[35] satirizing these healers, wrote:

> Faint was I only, Symmachus, 'til thou
> Backed by an hundred students, throng'dst my bed;
> An hundred icy fingers chilled my brow:
> I had no fever; now I'm nearly dead.

It may be inferred from Pliny that these quacks were largely foreigners: "The dignity of the Roman," wrote Pliny, "does not permit him to make a profession of medicine, and the few Romans who begin to study it are venal renegades to the Greeks." Pliny was against these foreign healers. "The Roman people," he stated, "in extending its empire, has lost sight of its ancient manners and, in that we have conquered, we are the conquered; for now we obey the natives of foreign lands, who, by the agency of a single art, have even outgeneraled our generals."

Many of these healers occupied themselves with diseases of the eye (*medici ocularii*). Most were in reality eye ointment venders. Rome of that period can boast of only one celebrated oculist, Demosthenes Philalethes, who flourished at the time of Nero. He wrote a textbook on diseases of the eye which was in use down to the fourteenth century.

The discovery of about two hundred and thirty ancient seals in the northern and western provinces of Italy show that the Roman oculists employed salves widely. The seals, found attached to jars of ointment, bear the name of the ointment, of the oculist who discovered it and of the manufacturer and state the purpose for which it was to be used together with the directions for use. One ointment consisted of zinc and calamine. Some depilatories and tweezers for pulling out hair, possibly for trichiasis, were also found.

One inscription on a seal reads, "The incense salve of T. Attius Divixtus against attacks of ophthalmia." (*T. Atti. Divixti. Dia. Libanu*). The sigma states, further, "To be ground up with an egg" (*Ad. Imp. Ex. Ovo*).

On at least one seal there is an inscription indicating that some oculists specialized in surgery. The inscription reads, *"medicus clinicus chirurgicus ocularius."*[36] Martial poked fun at the eye doctors in

35. Martial Selected Epigrams of Martial, translated by E. Post, Chicago, Ginn & Co., 1908, chap. 5.

36. Gossen, A. H.: The Physician in Ancient Rome, Ciba Symposia **1**:9 (May) 1939.

the following words: "Now you are a gladiator who once were an ophthalmist; you did as a doctor what you do as a gladiator." "The blear-eyed Hylas would have paid you sixpence, O Quintus; one eye is gone, he will still pay threepence; make haste and take it, brief is your chance; when he is blind, he will pay you nothing."

The oculists of Alexandria at various times throughout the period of the Roman Empire were remarkably skillful. Their literature has perished, but it is believed that they were able to operate for cataract.

A real advance in Roman medicine began with the arrival of Asclepiades, of Prusa in Bithynia, in the beginning of the last century B. C. He started out in Rome as a teacher of rhetoric, but on the advice of his friend Cicero he gave up teaching for the study of medicine. He became famous among the Romans because of his strong opposition to the Hippocratic doctrines. He opposed the humeral theory of Hippocrates and advocated in its stead the atomic theory of Democritus. His teaching led to the medical school of the methodists. In other matters he was a follower of the Epicurean philosophy. Asclepiades may be ranked as the first foreign physician who contributed to the establishment of Greek medicine in Rome. It was due to his follower Musa that Julius Caesar bestowed citizenship on Greek physicians. This decree, however, did not change the medical standards of the Roman physician in general, since there was no examination necessary to practice medicine. After Caesar's death following his military campaign in Egypt and Spain, his successor, Augustus, who was successfully cured by Antonius Musa from a severe inflammation of the liver, showed his appreciation to the medical profession in general by exempting all physicians from taxation, and as a personal token of appreciation he ordered the erection of a statue of Musa among the Caesars and presented him with a signet ring of knighthood.

From that time on there was a progressive medical development in Rome. Physicians became a class of citizens more or less protected by law, although they never attained the same dignity as in Greece. With the exception of court physicians, military surgeons and civilian state physicians, Greek practitioners were still looked on as captives or freedmen.

Not until the reign of Septimius Severus (reigned 193-211 A. D.) was medical practice definitely legalized. He introduced a system of licensure and made the medical practitioner responsible to the state for his actions. Criminal practices, such as the administration of abortifacients, were punished by fines and revocation of license.

The Romans were probably the first to experiment with socialized medicine. There were state, circuit and municipal physicians, as well as school physicians and physicians attending the vestals. The leading government physicians were known as *archiatri*, from which the German

name *(arzt)* is derived. The poor received free medical treatment. The number of physicians allotted to a community was fixed by law. Overcrowding of the profession was controlled by law: Antoninus Pius decreed that no more than ten physicians could be employed in the average large community. Smaller cities were permitted from five to ten physicians, according to the population. Rome, the capital city, had fourteen physicians besides one for the vestal virgins and one attached to the gymnasiums. These, although they received fixed salaries, were permitted to charge patients of the wealthier classes for medical services.

Neither time nor tide affects human nature. The fact that wealthy persons not infrequently feigned poverty in order to receive free medical attention will strike a familiar note with present day practitioners. Galen, who had a large private practice in Rome, complained of the annoying experiences he had with certain rich patients. They frequently deceived him in order to test his ability as a diagnostician. This medical genius, however, readily detected a liar by feeling his pulse.

In the age of Augustus, culture in Rome reached its highest level. This monarch, who established a constitutional form of government (27 B. C.) also introduced ordinances which improved the sanitary and hygienic conditions of the land. These ordinances were enforced by the archiatri.

Because of their importance to the welfare of the citizens, Augustus recognized the state physicians as a privileged class. He regulated the number of physicians employed in the army. Each military regiment had a staff of twenty-four surgeons. The North Sea naval squadron also had an ophthalmologist. These military medical practitioners were under the direct command of military officers and had no medical superior. In contrast to the civil physicians, who received fair remuneration for their services, military physicians were poorly paid. It is not clear to what extent malpractice was controlled by the Romans of that period. Judging from the steps taken to punish criminal abortion, it may be assumed that the practice of medicine was well regulated.

A great change took place when the Roman State was overthrown. The transfer of the world's capital to Constantinople dealt a death blow to medical science. There were no celebrated physicians for many years after the demise of Galen. No medical investigation and no original work worth mentioning was done. The greatest ambition of the physician was to be able to comment on the works of Hippocrates and Galen. Indeed, during the first four hundred years of the present era, medicine was altogether removed from the cultural environment and drawn into the narrow confines of the church, where it became dogmatic and was practiced largely by monks and the treatment depended mainly on faith.

The status of the physician, which usually rises and declines with the advancement and decline in medical arts and sciences, reverted to the rigid laws of ancient Babylonia. Thus, during the third and fourth centuries A. D. the Visigoths (a Teutonic people who overran Europe and settled in France and Spain) demanded that the physician make a pledge of cure before taking a patient. If the patient died the physician received no fee and the relatives of the deceased could deal with him as they saw fit. Injury to a nobleman during bloodletting carried a penalty of 100 *solidi* (equivalent to about $225). A dead or injured slave had to be replaced with one of equal value. No physician was allowed to visit a prisoner lest he defeat justice by furnishing him with poison.

In the early centuries of Christianity, medical jurisprudence was greatly affected by the Biblical teaching that disease is a divine affliction and particularly by the religious philosophy of predestination, or the determination of man's destiny beforehand by God. According to these concepts, the physician is only a messenger of God and cannot be responsible for injury or death occurring during the course of treatment. Josephus [37] stated that there was a division of opinion among the Hebrew religious philosophers of his time on this subject. The Essenes attributed all matters to divine predestination, denying to man any control in shaping his future. A later rabbi, expressing the Essenic thought, stated, "A man does not hurt his finger in this world, unless it has been decreed from above." [25] Another Talmudic rabbi declared, "A plague may rage for seven years and yet no man will die before the appointed hour." [38] On the other hand, the Sadducees, a liberal Jewish party of the same period, who followed the teaching of the Epicureans, held that all phenomena of the universe are due to chance; they denied the existence of personal divine providence. The third party, the Pharisees, adopted a compromise view, declaring that man's occurrences in life are predestined only with regard to material things but are completely undetermined with regard to spiritual matters. The latter view is expressed in the teaching of the sage Rabbi Akibah, "All is foreseen yet freedom is granted." He wished to convey the idea that man can choose between right and wrong.[39]

The idea of predestination in the wider sense was taught in the Christian church by St. Augustine but was opposed by other church fathers, who favored conditional predestination, which gives man the will to choose between right and wrong. With regard to medicine,

37. Josephus, F.: The Jewish War translated by W. Whiston, Boston, S. Walker, 1833, book 2, chaps. 8 and 11; footnote 18, book 15, chap. 10.

38. Talmud: Sanhedrin 29 *a*; Yebomoth 114 *b*.

39. Abboth: 3:15.

however, the prevailing opinion was that conditions of health and disease emanate entirely from divine sources: "'I wound and I heal,' sayeth the Lord."[40] "'I God am your healer.'"[41] A physician, therefore, was considered the messenger of God, whose duty was to carry out the will of the Lord. Penitence and prayer were the only means that could change the fate of a person if he was once marked to be sick. This stoic doctrine of "what has to be cannot be changed" was responsible for the fatalism among the followers of Islam, who became convinced that the processes of the world and of man cannot be changed by man. The old Leonite dictum "No medicine when the end is to come" was the general belief of the religious philosopher.

According to this view, the physician could not be held responsible for damage done to his patient in the course of treatment; he was merely a tool in the hands of the Lord. The only justification for penalizing the physician was for interfering with providence in attempting to treat sick or injured persons. If God wounds and heals, it was argued, the only persons that can act for providence are His servants, the priest and the monks, who know the ways of the Lord and can propitiate Him by offerings and prayers.

In the great discussion on predestination that was raging among the Islamic philosophers, Maimonides, the Jewish sage of Córdoba, combated the idea of predestination, stating that neither material nor spiritual affairs are predestined. He threw his authoritative weight on the side of the Islamic Mutazalites, who were opposed by the school of Ashari.[42]

From the fifth to the twelfth century, continuous strife went on between the representatives of the church, who claimed to be the only divinely chosen practitioners of medicine, and the lay physicians, who almost worshipped Galen as their patron saint. The difference between the two groups of contenders for the toga of Aesculapius was rather in the methods of treatment than in the philosophy of medicine, for the lay physicians were as a rule devout Christians. It must be remembered that Galen himself was a devoted monotheist who frequently sang songs of praise to the Lord for his wisdom in the construction of man. Just as many lay physicians were religious, so numerous monks who attended to the sick, particularly those of the Benedictine order, were admirers of Galenic medicine. In practice, however, the priests diluted the art of medicine with religion, magic and astrology, while the lay physicians practiced unadulterated Galenic medicine. Another difference was that the church occupied itself only with internal medicine. Its votaries were either monks or schoolmen. Some of the latter were fore-

40. Deuteronomy 32:39.

41. Exodus 15:26.

42. More Nebuchim 3, chap. 17.

most intellectual leaders, such as Thomas Aquinas, Roger Bacon and Albertus Magnus. The church was against surgery. It abhorred the shedding of blood. On the other hand, the secular healer favored the practice of surgery even though he risked his own life or limb as well as his patient's. In 1337 an oculist was drowned in the river Oder because his couching operation for cataract on John of Bohemia resulted in blindness.

Medical jurisprudence in the Middle Ages was of the crudest kind. Expert opinion was based on casuistry and hair-splitting arguments. The bleeding of a corpse in the presence of a suspect was sufficient to determine his guilt. The blood of a person who died by violence was believed in one way or another to bear witness against the guilty one, "for the blood of the dead does not rest until the guilty one is apprehended and punished."

As late as 1688, during the trial of Philip Standsfield for the murder of his father, Sir James, testimony was produced that when the son touched the dead body of his father the blood flowed afresh. Sir George McKenzie, sitting at the inquest, said that the jury was fully persuaded by the oozing of the blood that Sir James was murdered by his own son.

This gross ignorance of medical phenomena was first attacked in Italy, where the universities of Salerno and Montpellier were established and where medical guilds were formed to regulate the status of medicine. The liberal King Roger and his successors on the throne of Sicily greatly contributed to the organization of medical practice in Italy on a scientific basis.

In 1140, King Roger issued an edict that the physician could not practice medicine without proper examination, under the penalty of imprisonment and confiscation of property.

Eighty-four years later, his grandson, Fredrick II, enlightened by wisdom gained from the Orient, continued the good work of his grandfather. He promoted all cultural pursuits among his people but particularly the practice of medicine. He enacted laws to govern medical teachings and practices. He introduced a decree that before any one entered on the practice of medicine he had to pass an examination before the masters of Salerno covering the teachings of Hippocrates, Galen and Avicenna. The examination was given to candidates after they had pursued a course of study including three years of logic, five years of medicine and one year of "internship" under an experienced medical practitioner. The candidate for a license in surgery had to produce evidence that he had studied surgery for at least a year and that he was familiar with human anatomy. He was examined by three professors, one of whom conducted the examination in the Latin language. The medical aspirant had to produce testimonial

letters as to character before he was granted his certificate. A license was issued by Fredrick himself to those who passed a successful examination in medicine and surgery.

The physician was required to treat the poor free and to visit his patient twice a day and once a night if necessary. Collusion with the apothecaries was forbidden, and any substitution or adulteration of drugs had to be reported to the authorities. The sale of poisonous drugs, abortifacients and secret remedies was punishable by death if any person lost his life thereby. Periodically, inspectors visited the apothecaries and examined their mixtures. Sanitary and hygienic regulations were strictly enforced. The medical regulations enacted by Fredrick II and published in the year 1224 secured for him an honorable place in the history of medical culture.[43]

Whether prompted by the associated high mortality or merely by opposition toward "shedding of blood," the Council of Tours in 1163 A. D. prohibited clergymen and monks from performing any major operation. From that time on the clergy confined itself to prescribing medicine. As a result, the practice of surgery fell into disrepute and for a time it was almost entirely in the hands of barbers, who employed in front of their shops the familiar poles painted with red and white stripes. The red symbolized blood and the white represented bandages.

Incidentally, in England as late as 1540 A. D. guilds of barbers and surgeons were united by an act of Parliament. This dubious union existed in England until 1745, when the barbers were ousted, leaving the surgeons in complete control of the practice of surgery.

Legal regulation of medical practice in England began with the reign of Henry VIII. In the year 1511, by a statute signed by the English ruler, the practice of medicine was divided between physicians and surgeons and was placed under the jurisdiction of the clergy. This law reads as follows:

> No physician or surgeon shall practice in London, or within seven miles of it, without examination of the Bishop of London; or the dean of St. Paul's and four doctors of physic; nor out of the city or precinct but if he be first examined and approved by the Bishop of the Diocese or his vicar general calling to them such expert persons in the same faculty as their discretion shall think convenient.

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43. On this side of the Atlantic the first attempt to regulate medical practice was in the year 1649, when physicians were few and quacks were numerous. The following ordinance was passed: "For as much, as the law of God allows no man to impair the life or limbs of any other, but in a judicial way, it is, therefore, ordered that no person or persons whatsoever, employed at any time about the bodies of men, women, and children . . . presume to exercise or put forth any act contrary to the known approved rules of the art . . . nor exercise any force, violence, or cruelty upon or toward the body of any, whether young or old . . . without the advice and consent of such as are skilful in the same art (if such may be had), or at least some of the wisest and gravest there present, and consent of the patient or patients, if they be mentis compotes. . . ."

Surgery was declared a part of physic in 1560. Since the days of Henry VIII, numerous acts of Parliament have been passed affecting the medical profession. One enactment passed under the reign of James I prevented religious recusants from practicing physic or engaging in the art of the apothecary.

Throughout the ages, there has been a willingness or even an eagerness on the part of the patient to reward the healer for his services. At a time when medicine was still in a primitive state, connected with priestcraft and dispensed from the temples, votive offerings to the gods were a part of the cure. The walls of the temples were literally covered with gifts of gold, silver and precious stones. Many of these were fabricated in models resembling the part of the body affected by the disease.[44]

In Babylonia, the fees of the physician were regulated by law. The basis for determining the fee was the nature of the ailment, the rank of the patient and his ability to pay. Thus, if a physician opened an abscess of a freeman with a bronze lancet and cured his eye, he was to receive 10 shekels of silver; if the patient was poor, the doctor's fee was cut to 5 shekels; if the patient was a slave, his master paid only 2 shekels for the operation.

In ancient Egypt, it was customary to reward the healer with livestock. If the patient was the head of a clan or belonged to the well-to-do class, an entire estate or farm was not too great a reward for medical services. The average citizen's gift was a small ox. The fees presented to healers by wealthy women, such as the wives of chiefs of clans or of mayors of municipalities, differed from the payments made by rich men. The wife of the chief of a clan customarily paid the physician with a horse, while the wife of the head of a municipality or province paid with a she-camel.

In China, medical fees were regulated by law at an early date. The Chinese frequently expressed their gratefulness to their healers by issuing testimonials. They inscribed the names of their physicians on panels which were displayed in the temples, public places, bathhouses, etc. It is related that Emperor Kao Tsing (1127-1163 A. D.) honored Wang Futan, the physician who treated his mother for an ocular infection, by inscribing his name on a panel. Such a panel inscribed with the words for "purity" and "tranquility" decorated the emperor's temple.

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44. It may be of interest to note here that during the colonial days, doctors were paid with articles of barter. In New England they were paid with corn; in the Southern states, with tobacco. The Indians paid with wampum. In 1636, the assembly of Virginia enacted a law which provided that those who served an apprenticeship as surgeons and apothecaries should receive 5 shillings a visit and university graduates 10 shillings.

The Hindu physician, as has already been pointed out, did not need any state laws to regulate his professional fees. He possessed a strong ethical code of medical discipline deeply imbued with sagacity and earnestness that could not fail to make a favorable impression on his patient.

The early Greek physicians who practiced theurgic medicine in their temples received votive offerings from their grateful patients. These gifts consisted of models of parts of the body made of gold, silver and other precious metals, representing the seat of the disease. Patients also paid fees of standard coins. It is related that Aesculapius demanded gold and silver for his services and that one time he completely lost his divine dignity when he mumbled aloud to one of his patients, "Thou art healed now, pay the fee." In later Greece, when medicine became separated from religion, the fees of the physician were not legally defined. The high esteem in which the physicians of Athens were held assured their material rewards.

Among the Hebrews, the question of fees appears also to have been left to the patient and his physician, as may be inferred from the writings of Sirach:[45]

> Honor a physician with the honor due unto him, for the uses which ye may have of him, for the Lord hath created him . . . and he hath given men skill, that he might be honored in his marvelous work. . . . My son in thy sickness be not negligent. . . . Give place to the physician. . . . Let him not to go from thee for thou hast need of him.

The apocryphal books mention the case of Tobit, who paid an oculist of Media 5 talents of silver (a fee equivalent to $10) for the treatment of leukoma of the eye.[46] The physician continued to be held in high esteem in Syria down to the Roman period. According to the Syro-Roman law book: "If a man has been taken in hand by an *assia* (physician) and he gave his pay, the patient cannot recover the sum whether he is healed or not."[47] The same work prohibits blood vengeance and requires the accuser to hand over the guilty party to the authorities.

Among the Hebrews of the early centuries of the present era, the physician occupied a prominent place. It was considered hazardous to live in a place where there was no physician.[48] A circuit physician was employed in districts where single communities were unable to employ a physician. Physicians were attached to courts of justice to

45. Sirach 38:1, 2, 3 and 4.

46. Tobit 2:10.

47. Bruns and Sachu, cited by Cook, C. A.: Bible and Code of Hammurabi, London, Adams & Chas. Black, 1903, p. 38.

48. Talmud: Sanhedrin 17 *b*.

give expert testimony in criminal cases and to pass on the character of the injuries sustained in cases of assault. The court's judgment was based on their opinion.[49] When corporal punishment was decreed, a physician had to examine the prisoner to determine if he could safely endure it. A physician who offered his services free was not trusted: "A physician who takes nothing is worth nothing." [50]

The professional incomes of physicians varied as greatly in ancient Greece and Rome as they do at the present time. A few were paid very large fees, but the average physician's income in private practice was relatively low. Herodotus mentioned that the Aeginetans (in 532 B. C.) paid Democedes, from the public treasury, $1,570 a year [51]; Seleucus paid Erasistratus about $100,000 for curing his son Antiochus. The Athenians afterward paid him $2,030 a year, and at Samos he received $2,110 yearly. Quintus Stertinius earned about $35,000 yearly. Pliny said that Albutius, Arruntius, Calpetanus, Cassius and Rubrius each made close to $10,000 a year and that Quintus Stertinius favored the emperor by accepting about $20,000 a year when he could have made more in private practice. Pliny stated that Manlius Cornutus paid his physician $10,000 for curing him of a disease of the skin. The surgeon Alcon accumulated a fortune of nearly $500,000 during a few years' practice in Gaul. Galen, who had a large practice while he was in Rome, received about $1,750 for treating the wife of Consul Boetius.[52]

C. Stertinius Xenophon together with his brother Quintus left more than $1,760,000. The Arruntius mentioned is said to have bequeathed a still larger fortune. Horatillavus, a quack, who was lucky enough to bring about the cure of the poet Titus Leno, is said to have saved 150,000 *sesterces* in a few months. He accumulated a fortune of $6,000,000 practicing hydrotherapy, employing water at various temperatures. The large fees charged by certain physicians in the time of Pliny aroused the ire of the historian. His bitter complaint that physicians extracted huge sums of money from him led to the compilation of a prescription formulary called "Medicina Plinii." It was intended by the author as a sort of self-treating device. A century or more earlier, Cato had the same grievance against Greek physicians: "They demand expensive fees in order to make one trust them. In this way they can carry out their sinister game with greater ease."

The fees of the average physician, however, were not unduly large. The remuneration for a professional visit one century B. C. was

49. Talmud: Sanhedrin 18 *a*.

50. Talmud: Baba Kama 85 *a*.

51. Herodotus 3: 129-137.

52. Elliot, J. S.: Outline of Greek and Roman History, New York, William Wood & Company, 1914, p. 162.

approximately 65 cents or even less. It should be borne in mind that in the days of Caesar the purchasing power of money was twenty times greater than at present.

Military physicians under Caesar (102?-44 B. C.) were underpaid. They received only about $50 and board per annum. Under Domitian (reigned 81-96 A. D.) they received $66 and under the liberal Septimius Severus about $110.

Generally speaking, physicians' fees in ancient Rome were more liberal than in twelfth century Italy and certainly more generous than in sixteenth century Germany. The fee for an office or local call under Fredrick II was stipulated to be half a *tarenus* (about 35 cents). For out of town visits a fee of about $3 included the physician's traveling expenses, while $2.50 was the sum paid if the patient took care of the traveling expense. In sixteenth century Germany the pay given to the physician was still less. A single visit rated from 8 to 50 cents. Consultants, however, were fairly paid; the fee was $2.50 for a personal consultation and $1.25 for advice given by letter. Municipal physicians received from $4.25 to $43 annually; court physicians got from $35 to $939.

The physicians who attended Henry II, Henry III and Queen Elizabeth received $200 annually. Certain Roman physicians during the first 500 years A. D. demanded large fees even from the clergy. The Pope was no exception. A contemporary physician of Pope Honorius IV demanded 400 ducats for each day's attention. The Pope promised only 100 florins but gave him 1,000 on recovery. The largest fee on record paid to an obstetrician was that given by Catherine de Medici to Jean Fernel. The amount was $10,000 for each delivery. The best paying specialty appears to have been that of genitourinary diseases. The treatment of syphilis and gonorrhea fetched large sums of money. Casanova in his memoirs conveyed a fair idea of the prevalence of venereal diseases of his day and the readiness of those afflicted with them to pay the physician for professional services.

The caliphs liberally rewarded physicians. A certain enterprising medical man, Gabriel Bachticula, is said to have received $125,000 from a caliph in a single fee. He was also paid $560 monthly and received beneficent gifts each new year's day. Incidentally, the Mohammedans appear to have been most lenient with their healers in cases of malpractice. Puffindorf told of an Arab who called in a *farrier* (veterinary) to attend to his eyes. As a result of the veterinary's using remedies customarily given to quadrupeds, the Arab lost his sight. The judge before whom the case was tried decided against the plaintiff, remarking, "If the patient had not been an ass, he would never have employed a horse doctor."

It is apparent that the regulations governing medical practice were always in direct proportion to the value placed on human life and that this in turn was closely related to the degree of cultural advancement. The ancient Babylonian culture cast a magic spell over the minds of man yet made him conscious of his destiny with reference to law and order. The Greek culture gave birth to scientific medicine but was not practical enough to legalize it. The Hebrew culture was founded on the principle that all phenomena of life are governed by a supreme being; yet it permitted a physician to meddle in the affairs of providence. The Roman culture was built up by the sword; yet justice and law became its heritage.

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TRANSPLANTATION OF SUPERIOR OBLIQUE MUSCLE FOR OCULOMOTOR NERVE PARALYSIS

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Fourteen years ago Meyer Wiener [1] described an ingenious procedure to correct the pronounced divergence resulting from third nerve paralysis. As he stated, Edward Jackson and Dransart had, unknown to him, suggested a similar procedure and performed it on cadavers but not on patients with third nerve paralysis. Wiener's procedure included weakening the external rectus and transplanting the superior oblique muscle to an attachment beneath the insertion of the internal rectus muscle. He approached the superior oblique on the globe after cutting the superior rectus muscle, which was later replaced. The tendinous portion of the muscle was followed to the pulley, and with a scalpel the pulley was cut, the muscle being freed. The tendon was severed from the globe and the muscle dissected back into the orbit. About ½ inch (1.3 cm.) of tendon was cut off, and the end was sutured beneath the insertion of the internal rectus muscle.

In his original paper Wiener reported the results of operation in 2 cases. In the first case 15 degrees of divergence and 10 degrees of sursumvergence remained in the primary position, but the eye could be adducted 10 degrees past the midline. A later shortening and reattachment upward of the transplanted muscle were proposed. In the second case the muscle was sutured above the insertion of the internal rectus muscle, and as a result no sursumvergence developed. The effect on the divergence was insufficient, however, and at a second operation the transplanted muscle was further shortened and advanced to a point nearer the limbus. The result was parallelism in the primary position with power of slight inward rotation.

In a later article [2] and in his book on surgery,[3] Wiener described the use of a Weber knife to sever the pulley and exposure of the superior oblique muscle as it passes under the superior rectus without a division of that muscle. He also advised resecting more of the superior oblique

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1. Wiener, M.: Arch. Ophth. **57**:597, 1928.
2. Wiener, M.: Surg., Gynec. & Obst. **62**:487, 1936.
3. Wiener, M., and Alvis, B. Y.: Surgery of the Eye, Philadelphia, W. B. Saunders Company, 1939, p. 396.

and suturing it just above the insertion of the internal rectus muscle, to avoid a sursumvergence, as produced in his first case. In a personal communication, he reported having performed the operation in 2 additional cases with these changes in technic and stated that his associate Dr. Alvis had operated in a third case, good results being obtained in all cases. In spite of the fact that the muscle was inserted above the insertion of the internal rectus muscle, the tendency to sursumvergence previously noted was encountered.

In 1933 Peter,[4] apparently without knowledge of Wiener's work or of the suggestions of Jackson and Dransart, reported a case in which a similar procedure was employed. The case was one in which a stab wound of the orbit resulted in paralysis of the inferior rectus, the inferior oblique and the internal rectus muscle, the superior rectus being spared. The operative procedures included recession of the external rectus, tucking of the internal rectus and transplantation of the superior oblique muscle, all operations being performed in one session with the patient under general anesthesia. In transplantation of the superior oblique muscle, the tendon was exposed back of the pulley through a cutaneous incision. The reflected portion was grasped and cut between the pulley and its insertion on the globe. The pulley was opened and the tendon freed from it, a suture being placed in the cut end. Through an opening in Tenon's capsule over the internal rectus muscle, the suture was grasped and the muscle was pulled through and sutured to the attachment of the internal rectus muscle. The excess length of tendon was cut off in front of the suture. After a month lateral rotation was good, but the eye was elevated by the intact superior rectus. This muscle was then transplanted 3 mm. back and 3 mm. to the temporal side of its old insertion, and a Hunt-Tansley operation was performed to raise the upper lid. The result was parallelism in the primary position with a useful field of binocular vision and adduction of 30 to 40 degrees. Diplopia in parts of the field in which deviation was present was not noticed by the patient, a boy of 13, who learned to suppress or to avoid these portions of the field. Peter advised against the operation for adults with good vision in the paralyzed eye, on account of probable diplopia.

The present report concerns the results of operation in 5 of my cases with notes on a sixth case furnished by Dr. William Stokes and Dr. Harold Gifford Jr., of Omaha. Transplantation of the superior oblique muscle was chosen because of failure of other methods to effect permanent cosmetic improvement in this disfiguring condition. Resection and advancement of the paretic internal rectus, with recession or tenotomy of the external rectus muscle, had, in my experience, produced temporary improvement, but contracture of the unopposed external rectus

4. Peter, L. C.: Tr. Am. Ophth. Soc. **31**:232, 1933.

had always occurred later with return to a condition resembling the original one. Such a result had occurred in Wiener's first case a year before the transplantation was performed. In a case of my own in which the superior and the inferior rectus muscle had apparently recovered after what was originally a complete third nerve paralysis, transplantation of the inner half of the superior and of the inferior rectus muscle beneath the insertion of the internal rectus, with recession of the external rectus, was performed, but the result was far from satisfactory. It seemed that, although some function had returned to the vertical rectus muscles, there was still not enough active innervation to make their transplanted halves useful. Cases chosen for the Wiener operation were instances of third nerve paralysis in which the ptosis had completely or in large part disappeared. Ptosis was still present in case 6. In all cases pronounced divergence was present in the primary position with, in all except case 5, inability to adduct the eye past the midline.

The technic employed resembled that described by Peter, as it seemed simpler to reach the trochlea by a direct incision. It must be admitted that this exposure is the most difficult part of the operation, involving considerable dissection in a vascular field. It was possible, however, in all cases to identify the muscle by movements of the globe when a hook was passed beneath its reflected portion and to dissect it from the pulley under direct observation. The scar of the cutaneous incision was in most cases invisible. It seemed important to free the muscle far back into the orbit, so that it could be made to pass as nearly in the direction of the internal rectus muscle as possible in its transplanted position. It also seemed important to make the opening in Tenon's capsule directly over the internal rectus muscle and not above it, as was done in 1 case. The length of tendon to be cut off was estimated by holding the muscle over the cornea while the eye was held in a slightly overcorrected position, recession of the external rectus muscle having first been completed. The muscle clamp was placed at the point where the muscle crossed the scleral groove prepared for it 3 mm. from the corneal limbus, and the muscle was cut off in front of the clamp. Tucking or resection of the internal rectus muscle was not employed except in case 1, and the muscle was left intact. A firm attachment for the muscle was afforded by a groove dissected obliquely in the superficial scleral layers, a method employed for advancement in my cases during the past ten years. Two sutures with an extra loop around the peripheral fibers, as employed in my advancements, were used. In some cases the tendon at the point where it was cut off was not flat, as is the tendon of a rectus muscle, but rather round and solid, like a segment of rope. Removal of as much capsule as possible from the tendon made it flatter, so that the resulting lump beneath the conjunctiva was less noticeable. In all but 2 cases general anesthesia was employed. In these cases, however, retrobulbar injection,

with infiltration in the region of the pulley, afforded satisfactory anesthesia.

CASE 1.—L. W., a woman aged 41, had extreme divergence of the right eye resulting from oculomotor nerve paralysis due to syphilis. The ptosis had cleared up, but the right eye was held in extreme abduction and could not be adducted farther than a point 35 degrees out from the midline. Wiener's procedure was performed, the right superior oblique muscle being separated from the pulley, cut in front of its insertion on the globe, 1.5 cm. of it resected and the stump sutured to the sclera 2.5 mm. in front of the insertion of the internal rectus muscle. Tucking of the paretic internal rectus and a 5 mm. recession of the external rectus muscle were also performed. Recession and advancement of the paretic inferior rectus was later performed to aid a sursumvergence which developed after the first operation. There was some improvement, but the right eye was still above the left.

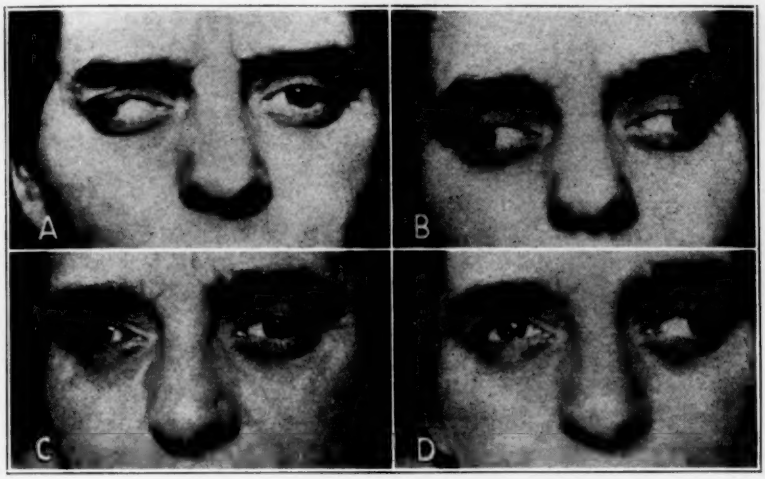

Fig. 1 (case 1).—*A*, before operation, primary position; *B*, before operation, eyes left; *C*, after operation, primary position; *D*, after operation, eyes left.

At a third operation the transplanted muscle was exposed. It was found firmly adherent to the sclera slightly above the insertion of the internal rectus muscle. It was freed from a rather broad attachment and from fascial attachments as far back in the orbit as possible. Five millimeters was resected, and it was sutured carefully in a scleral groove 2 mm. in front of the insertion of the internal rectus muscle, an attempt being made to obtain a pull from the muscle in a straight lateral direction. The external rectus muscle, which was found attached near the equator, was tenotomized. This procedure improved the position of the eye considerably. It was only slightly sursumvergent, and the primary divergence was reduced from a previous 70 degrees to 30 degrees. When the patient looked straight to the left it could be moved to the midline, but not past it. When the patient looked down and to the left the right eye was adducted 5 to 10 degrees past the midline (fig. 1). Five years later the divergence had somewhat increased, but the cosmetic improvement over the original condition was still considerable.

CASE 2.—M. B., a girl aged 15, showed a paralysis of the right oculomotor nerve which was complete except that the ptosis was only partial. The condition followed an injury to the head at the age of 5. The right eye was divergent 60 degrees on the perimeter in the primary position, and movements were limited to a small area up and out from the fixation point. The eye could not be adducted to the midline. Operation was performed at Cook County Hospital according to the procedure previously outlined, which included recession of the right external rectus and transplantation of the right superior oblique muscle to a point just in front of the insertion of the internal rectus. The patient disappeared from observation not long after operation, but at that time the cosmetic improvement was pronounced. The lid drooped for a time after operation, but this ptosis improved within two weeks. The right eye was divergent only 5 to 10 degrees in the primary position and could be adducted 10 degrees in from the midline. As in case 1, some sursumvergence was present, but it was considerably less, not over 10 degrees. Procedures to correct this were considered, but the patient did not return.

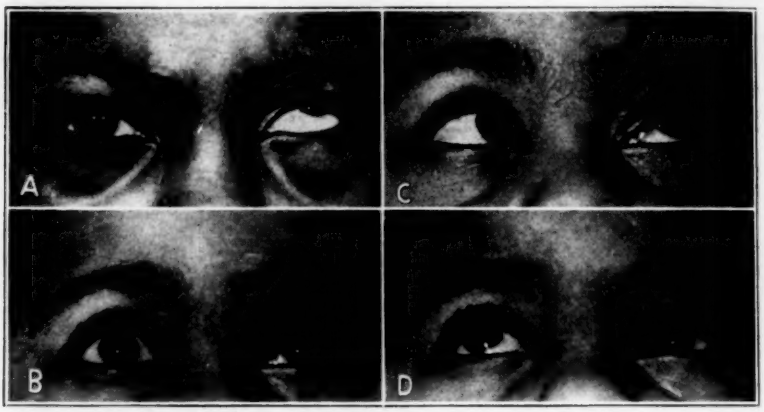

Fig. 2 (case 4).—*A*, before operation, the patient fixing with the right eye; *B*, four months after operation, primary position; *C*, four months after operation, eyes left; *D*, four months after operation, eyes right.

CASE 3.—K. K., a youth of 19, showed a paralysis of the left oculomotor nerve which had been present since birth. The left eye diverged 50 degrees on the perimeter in the primary position and on extreme adduction remained 30 degrees out from the midline. It turned down on attempted adduction owing to action of the superior oblique muscle. Slight ptosis was present. Operation July 28, 1941 included free tenotomy of the left external rectus and transplantation of the left superior oblique muscle to a groove 3 mm. in front of the insertion of the internal rectus.

Three weeks after operation the left eye was straight in the primary position with no gross vertical deviation. Slight left sursumvergence could be shown on cover test. On looking to the right the left eye was adducted 15 degrees in from the midline, but the unaffected right eye was rotated much farther to the right in all parts of the right field. Fusion tests were not made, but there seemed to be binocular central vision in a small central field. Although vision in the left eye was 20/50, the patient did not complain of diplopia.

CASE 4.—J. T., a Negro girl aged 5, had shown a left divergent squint since birth. There was no history of an injury at birth. Examination showed left divergence of 20 degrees and left sursumvergence of 10 degrees. On looking to the right the left eye did not pass the midline and turned farther down. There was no upward movement of the eye. On the patient's fixing with the left eye the right eye showed marked secondary divergence up and out. Vision was 10/10 in the right and 4/10 in the left eye.

Operation included a 5 mm. recession of the left external rectus and transplantation of the left superior oblique muscle to a groove slightly above the midline

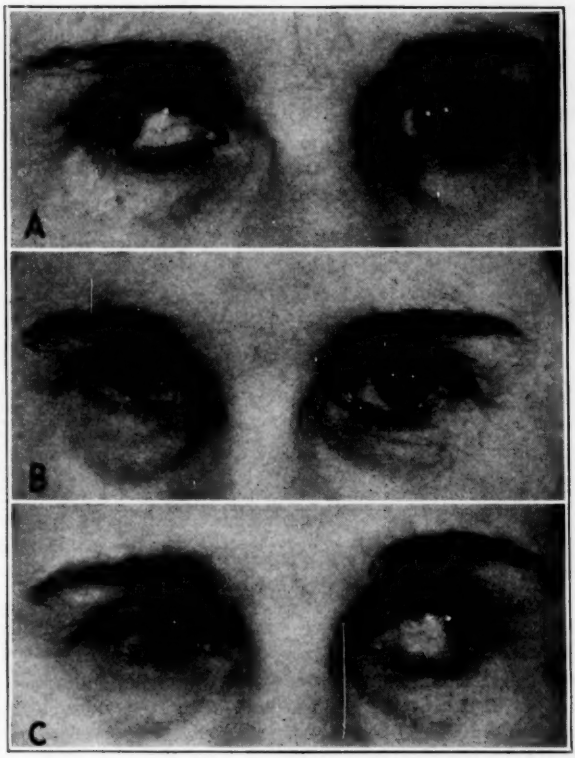

Fig. 3 (case 5).—*A*, before operation; *B*, three months after operation, primary position; *C*, three months after operation, the patient looking to the left.

and 2 mm. in front of the insertion of the internal rectus. The cosmetic result was excellent (fig. 2). The drooping of the lid which immediately followed the operation cleared up fairly well within three weeks. The left eye appeared straight in the primary position and could be adducted 35 degrees past the midline. No sursumvergence was present in the primary position, but on looking to the left the eye turned up 15 degrees, while abduction was distinctly limited.

CASE 5.—In Mrs. S., aged 41, right-sided oculomotor nerve paralysis developed in 1921, associated with right-sided facial nerve paralysis. When she was first seen, in 1941, vision was 20/15 in each eye. The right eye showed 60 degrees of divergence

on the perimeter in the primary position, with slight sursumvergence. On looking to the left the eye could be adducted 15 degrees past the midline and turned down owing to the action of the superior oblique muscle. Both the superior and the inferior rectus muscle had some function, as movement up and down of 20 degrees was possible. Abduction was normal. The ptosis originally present had cleared up completely.

Operation included 5.5 mm. recession of the right external rectus and transplantation of the right superior oblique muscle to a groove 2 mm. in front of the insertion of the internal rectus muscle. The operation was performed with procaine hydrochloride anesthesia, which proved satisfactory. When the sutures in the superior oblique muscle were tied the eye showed rather marked sursumvergence; so a tension suture was placed through the inferior rectus tendon and out through the lower orbital margin. This suture, however, did not prevent the rather marked sursumvergence from persisting. After four weeks the lateral position of the eye was excellent, and the eye could be adducted 15 degrees past the midline.

The superior rectus muscle was exposed. It was inserted 8 mm. from the limbus and was entirely flaccid; so it was not disturbed. The inferior rectus muscle was

Fig. 4 (case 6).—After operation.

also lacking in tone. It was dissected free from scar tissue, 5 mm. was resected and the cut end was inserted in a groove 3 mm. from the limbus. The result was a pronounced cosmetic improvement (fig. 3). The lateral position of the eye was satisfactory, and no gross sursumvergence was apparent. On cover test a slight sursumvergence was revealed. The ptosis had cleared up almost completely, but the palpebral fissure was slightly narrowed at the outer angle owing to drawing up of the lower lid. Adduction of 15 degrees was possible. Abduction was limited to 10 to 15 degrees.

Case 6.—Notes on this case were furnished by Dr. William Stokes and Dr. Harold Gifford Jr. H. D. had been seen by Dr. Harold Gifford Sr. in 1907, when he was 31 years old. He had suffered a complete paralysis of the right oculomotor nerve two months before. The history was interesting in that transient paralysis of the extraocular muscle had occurred on numerous occasions during thirteen years before he was seen, each attack being associated with severe headache and the picture being that of so-called ophthalmoplegic migraine. After the last attack the condition persisted, all muscles supplied by the third nerve, including the levator, being completely paralyzed. When the patient was seen in 1935, he had lost almost all vision in the left eye as a result of recurring retinal hemorrhage. The right eye was unaffected by the latter con-

dition, having vision of 20/30, but was in extreme divergence and covered by the ptotic lid.

To facilitate use of the right eye, surgical intervention was undertaken. After free tenotomy of the external rectus, the superior oblique muscle was approached as previously described, cut half way between the pulley and its insertion and the cut end sutured beneath the insertion of the internal rectus. Later the lid was raised by a Machek operation. The result was a pronounced cosmetic and functional improvement (fig. 4). There was divergence of 10 degrees but no sursumvergence, and the eye could be turned in 5 degrees past the midline. Eighteen months later the condition remained the same.

COMMENT

Experience with this operation has only confirmed my opinion that it is the method of choice in the treatment of oculomotor nerve paralysis under circumstances making any operative procedure advisable. Naturally it should not be advised until enough time has elapsed to be sure that improvement in the condition will not occur, usually a period of at least one year. It will not be considered as a rule for elderly persons to whom the cosmetic defect is not important. When ptosis is complete and persistent, there may be some doubt as to the advisability of operation, but most young persons will prefer two operations to the deformity of complete ptosis. Peter's fear of diplopia if the operation is performed on adults does not seem justified, since in none of the present cases has this proved a serious inconvenience.

When oculomotor nerve paralysis is complete, certainly no other operation affords the possibility of supplying a living and innervated muscle as an adductor. Hence it is difficult to understand why no other cases have been reported aside from those of Wiener and Peter.

Which method of approaching the superior oblique muscle is preferable must be left an open question. I am encouraged to try Wiener's approach entirely through the conjunctival sac in the next suitable case.[5] The direct approach to the pulley, however, is not difficult and is always possible. In judging the amount of superior oblique muscle which should

5. Recently 2 additional patients have been operated on by Wiener's method. In 1 of these an attempt was made to free the superior oblique muscle through the opening in Tenon's capsule as Wiener suggested. This proved impossible, and the usual approach to the trochlea was made through the skin. The result was a considerable improvement in the position of the eye but little adduction.

The second case was of especial interest. The patient was an infant of 3 years in whom paralysis was limited to the inferior oblique and internal rectus muscles of the right eye. On looking to the left the right eye turned straight down at least 40 degrees owing to overaction of the superior oblique muscle. The Wiener procedure alone was employed without interference with the external rectus muscle, the shortened superior oblique being attached above the insertion of the internal rectus. The operation was performed by Dr. Dean Spear at Cook County Hospital, Chicago. The result has been excellent. The eye is straight in the midline; the downward deviation on adduction is entirely absent, and a surprising amount of adduction is possible on looking to the left.

be resected, it seems advisable to hold the eye in a slightly overcorrected position and to clamp the muscle at the point where it crosses the scleral groove prepared for it, cutting off the excess of tendon in front of the clamp. This is to be done after recession or tenotomy of the external rectus muscle. Which of these two procedures shall be preferred will depend to some extent on the amount of divergence present. Recession has as a rule proved sufficient, and, in spite of the amount of recession having been limited to 5 mm. in some cases, considerable limitation of abduction has resulted. This, however, seems to be a disadvantage insufficient to offset the great cosmetic improvement obtained.

Sursumvergence resulting from an upward pull of the transplanted muscle presents a problem which has not been met with perfect satisfaction. It seems best to place the muscle, not in front of the insertion of the internal rectus muscle, but, as suggested by Wiener, just above it and 2.5 to 3 mm. from the limbus, in a scleral groove. To do so must cause torsion of the globe, one would think, but this has not apparently caused subjective discomfort. If sursumvergence occurs, it may be improved by resection and advancement of the paretic inferior rectus muscle.

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RETINOPATHY IN JUVENILE DIABETES MELLITUS

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In 1856 Jaeger [1] described hemorrhages associated with yellowish, round and oval areas in the retina of a diabetic patient. These findings were confirmed and described as typical of diabetic retinitis. In 1891 Hirschberg [2] summarized an exhaustive study of this disease and distinguished three different types:

1. A characteristic involvement of the posterior pole of the retina with small, light, silvery areas and tiny hemorrhagic spots (retinitis centralis punctata diabetica).

2. Hemorrhages in the retina with secondary inflammatory changes (retinitis haemorrhagica diabetica).

3. Rare types of retinal changes which probably were not directly caused by diabetes.

In all these types the optic nerve was normal and a macular star figure was absent. This classification was generally recognized as correct, and Leber [3] had little to add in his standard work on diseases of the retina.

It was, therefore, revolutionary when Volhard [4] in 1921 stated that he never saw a patient with diabetic retinitis without hypertensive changes. There followed an extensive controversy in which the name diabetic retinitis was rightfully changed to diabetic retinopathy. Some authors, like Wagener and Wilder [5] in 1921, Friedenwald [6] in 1930 and Gresser [7] in 1933, denied the existence of a separate diabetic retinopathy at all. The confusion was so far reaching that Lichtwitz [8] in volume 7 of Schieck and Brückner's short handbook denied the exis-

From the service of Dr. E. B. Gresser, Department of Ophthalmology, Beth Israel Hospital.

1. Jaeger, E.: Beiträge zur Pathologie des Auges, Vienna, k.-k. Hof- und Staatsdruckerei, 1855, p. 33, table 12.

2. Hirschberg, J.: München. med. Wchnschr. **51**:521, 1891.

3. Leber, T., in Graefe, A., and Saemisch, E. T.: Handbuch der gesamten Augenheilkunde, Leipzig, Wilhelm Engelmann, 1916, vol. 7, p. 970.

4. Volhard, F.: Verhandl. d. deutsch. Gesellsch. f. inn. Med. **33**:335, 1921.

5. Wagener, H. P., and Wilder, R. M.: Retinitis of Diabetes Mellitus, J. A. M. A. **76**:515 (Feb. 19) 1921.

6. Friedenwald, H.: Tr. Ophth. Soc. U. Kingdom **50**:452, 1930.

7. Gresser, E. B.: Am. J. Ophth. **16**:612, 1933.

8. Lichtwitz, L., in Schieck, F., and Brückner, A.: Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1930, vol. 5, p. 450.

tence of a diabetic retinopathy and Schieck [9] in volume 5 upheld the traditional diabetic retinitis. One of the main points in favor of the vascular theory is the fact that the fundus changes are rarely seen in the juvenile form of diabetes.

There are two factors which make the rarity of rentinal changes in juvenile diabetes easy to understand: first, diabetes usually occurs in persons at or beyond middle age, and, secondly, in the preinsulin era the prognosis for young patients with diabetes was so poor that most of them died before an opportunity for retinal changes to develop could occur.

Duke-Elder [10] stated that patients under 40 years of age are exceptional, and Foster Moore,[11] whose youngest patient was 39, concluded that diabetic retinitis does not occur under the age of 35. The first description of fundus changes in diabetes, by Jaeger,[1] referred to a 22 year old patient, but study of the plates and of the case history make it apparent that Jaeger was dealing with diabetes complicated by hypertension or vice versa. Nettleship [12] found that among 59 of his own patients and 23 reported on in the literature the youngest was 35.

Kako,[13] analyzing 57 cases reported by Uhthoff, found 3 patients with retinal changes in the age group between 31 and 40 years.

In 1932 Adams [14] reported on a 22 year old man who had yellowish areas of retinitis and hemorrhages in both eyes. The blood pressure was 100 systolic and 75 diastolic. The urine had a specific gravity of 1.015 and showed 2.2 mg. per hundred cubic centimeters of sugar.

In 1936 Braun [15] mentioned a patient of 23 and another of 26 with the typical picture of diabetic retinopathy. Both had a normal blood pressure. The 23 years old patient had some albumin and a few red blood corpuscles and casts in her urine; the other had normal renal function.

In 1938 Heinsius [16] analyzed the ocular findings of 221 diabetic patients. He found 2 with diabetic fundus changes in the group under 40 years of age. The first was a 37 year old laborer, known to have serious diabetes of one and one-half years' duration, who had many spotlike and flame-shaped hemorrhages with some white areas in the

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9. Schieck, F., in Schieck, F., and Brückner, A.: Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1932, vol. 7, p. 1.

10. Duke-Elder, W. S.: Text-Book of Ophthalmology, St. Louis, C. V. Mosby Company, 1941, vol. 3, p. 2732.

11. Moore, R. F.: Medical Ophthalmology, ed. 2, London, J. & A. Churchill, 1925, p. 165.

12. Nettleship: Roy. London Ophth. Hosp. Rep. **15**:339, 1903.

13. Kako: Klin. Monatsbl. f. Augenh. **41**:253, 1903.

14. Adams, P. H.: Brit. J. Ophth. **16**:38, 1932.

15. Braun, R., in discussion on papers of Mylius and Braun, Ber. ü. d. Verhandl. deutsch. ophth. Gesellsch. **51**:164, 1936.

16. Heinsius, E.: Klin. Monatsbl. f. Augenh. **100**:207, 1938.

posterior pole of each eye. He had a blood sugar content of 273 mg. per hundred cubic centimeters, a blood pressure of 135 systolic and 90 diastolic and no renal pathologic change.

The second patient was a 17 year old secretary whose diabetes was diagnosed just before her admission to the hospital. The retina of each eye showed a whitish flame-shaped area above the macula and a fine pointlike hemorrhage near the margin of the left disk. The fundus changes cleared up after a three weeks' stay in the hospital. She had normal renal function. The blood pressure was 125 systolic and 90 diastolic.

In 1938 Boulin and Kaufmann [17] reported 2 cases of diabetic retinopathy in young persons. The first patient was a 33 year old man in whom diabetes was discovered nine months previously, after he had lost 37 pounds (17 Kg.). The urine contained a high amount of sugar but no albumin. The blood pressure was 130 systolic and 80 diastolic. Ambard's constant was 0.076. The fundus showed many hemorrhages and exudates in the posterior pole. Vision was reduced to light perception in the right eye and to 0.3 in the left eye. The second patient was a 35 year old woman with diabetes of one year's duration who was brought to the hospital in a comatose condition. The fundi showed paramacular exudates and exudates above the vessels. There were no hemorrhages and no edema. The blood pressure was 140 systolic and 80 diastolic. There was no albumin. The urea content of the urine was 0.3 Gm. per hundred cubic centimeters. Ambard's constant was 0.076.

In 1941 McKee [18] analyzed 2,360 cases of diabetes mellitus from the ophthalmologic point of view. He found 476 patients with retinal lesions related to their diabetes, and 4 of these were between the ages of 6 and 31 years. He stated:

> One of these had two small retinal hemorrhages; in the second there were numerous hemorrhages throughout the retina, and in the third, a woman aged 23 years, each fundus showed numerous small hemorrhages scattered over the whole retina, with here and there characteristic chalk white patches. The fourth patient, a woman aged 31 years, showed definite hypertensive retinitis with papilledema.

There are unfortunately no details as to the blood pressure, duration of the diabetes, etc., in this report.

REPORT OF CASES

CASE 1.—A 26 year old white book keeper, H. L., came to the clinic of Beth Israel Hospital complaining that she had had difficulty in near vision for the past few weeks.

17. Boulin, R., and Kaufmann: Presse méd. **46**:387, 1938.

18. McKee, S. H.: Fundus Oculi in Diabetes Mellitus, Arch. Ophth. **25**:773 (May) 1941.

Her father died in 1919 of influenza. Her mother and one sister were living and well. There were no metabolic, circulatory or allergic diseases in the family to her knowledge. As a child she had measles, chickenpox and whooping cough. Her tonsils were removed seven years before. Occasionally after eating tomatoes, nuts or whole wheat bread she had hives. At the age of 9 she began to lose weight. Her appetite was good, and she had a particular craving for potatoes and bread. It was noted that she was drinking large amounts of water. She was admitted to a hospital, where the diagnosis of diabetes was made. She was discharged as stabilized with 50 units of insulin per day. At the age of 12 she had her only episode of diabetic ketosis. During adolescence she had several attacks of hypoglycemia. During the preceding five years she never had any sugar determination and regulated her dose of insulin by her feeling of well-being. During this period she lost more than 50 pounds (23 Kg.). She also had a normal pregnancy. On discharge from the hospital after her delivery she was on a diet of 300 calories of carbohydrate, 80 calories of protein and 100 calories of fat, with 50 units of protamine zinc insulin every morning.

The physical examination gave essentially negative results. The blood count gave normal results (red cells 4,240,000 and white cells 8,050). The hemoglobin content was 86 per cent. Tests of the blood chemistry showed a urea nitrogen content of 8 mg. and a sugar content of 211 mg. per hundred cubic centimeters. The urine showed on three examinations no albumin, no casts, no red blood cells, 2 or 3 white cells and a varying amount of sugar. The blood pressure was 125 systolic and 90 diastolic.

The palpebral fissures of the two eyes were equal. The lids were normal. There was no exophthalmos or enophthalmos. The movements were normal and there was no nystagmus. The conjunctiva of each eye was slightly injected, but there was no ciliary injection. The corneas were normal. The grayish irises seemed to be normal. The pupils were round and situated in the center of the iris. They were equal and reacted to light and convergence. The lenses were clear. The vitreous was clear. The disk of each eye was clearly defined, was of normal color and showed normal vessels. In each fundus there were about twelve small round hemorrhages. They were irregularly distributed above and below the macular region. In the right eye two irregularly shaped white areas were visible above the superior macular vein. The vessels did not show any irregularities in caliber or any other signs of hypertensive or arteriosclerotic changes. The tension was normal. The peripheral and central visual fields were normal. Vision was 20/20 in each eye. There were slight hyperopia and impairment of accommodation.

The second case is mentioned because the patient came under observation for fundus changes when she was on the borderline of juvenile diabetes.

CASE 2.—L. F., a 37 year old woman, had diabetes of fifteen years' duration and took 90 units of insulin daily. Her blood sugar was under control. The urine did not show any albumin or casts but showed a varying amount of sugar. The blood pressure was never higher than 135 systolic and 80 diastolic. Examination of the eyes at her first examination showed a normal left eye and hemorrhages with white areas in the upper and the lower macular region of the right eye. In the course of several years of observation the left eye became similarly affected and retinitis proliferans developed in both eyes.

COMMENT

The controversial question as to whether there is a diabetic retinopathy proper or the retinal changes are secondary to hypertension or arteriosclerosis can be decided on three bases:

1. Clinical observation and experience in differentiating the fundus picture. There is a marked difference between the fundus changes in diabetes, in arteriosclerosis and in hypertension, especially with reference to the optic nerve and the macular region.

2. Study of the pathologic changes in eyes from persons known to have had diabetes who have come to autopsy. Koyanagi [19] examined histologically the eyes of a patient who was carefully observed during his lifetime and proved conclusively that in this patient the retinal changes had developed as an independent disease.

3. Reports of cases of retinal changes in diabetic persons, especially young patients, whose renal and blood vascular systems were normal.

It is possible to evaluate the varying views only after careful descriptions of many cases. The criteria for the recognition of sclerotic and hypertensive changes in retinal blood vessels are sufficiently established to show that many previous case reports are valueless because of their lack of description and that a new series of juvenile diabetic patients should be observed.

SUMMARY

Two cases of retinal changes in juvenile diabetes are reported. There was no renal pathologic change or abnormal blood pressure. There are a number of cases in the literature which suggest that diabetes itself may produce the retinal disease known as diabetic retinitis or diabetic retinopathy.

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19. Koyanagi, Y.: Klin. Monatsbl. f. Augenh. **94**:353, 1935.

PARTICLES OF STEEL WITHIN THE GLOBE OF THE EYE

CAUSE, DIAGNOSIS, PREVENTION, TECHNIC OF REMOVAL AND RESULTS

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In my consideration of intraocular foreign bodies, I will confine my remarks to particles of steel within the globe of the eye. I will not discuss particles outside the globe, whether or not they penetrate the conjunctiva, or particles which have penetrated the globe and lodged in the orbit. I will include particles which have partially penetrated the sclera or the cornea.

As a matter of fact, particles of steel within the globe constitute 98 per cent of all intraocular foreign bodies. The remaining 1 or 2 per cent are either glass, copper or nonmagnetic metals. Because these particles are nonmagnetic, their removal is most hazardous and at times impossible.

Among more than 1,800 intraocular foreign bodies, I have encountered only 10 which were not steel; 3 of these were copper, 3 were glass, 3 were lead (bullets) and 1 was a particle of stone or rock.[1]

ORIGIN AND CHARACTERISTICS OF THE BODIES

In dealing with this particular form of ocular injury it is important, particularly from a diagnostic standpoint, to know where the particles of steel originate and what physical force causes them to penetrate the coat of the eyeball. Practically all of these metallic bodies are of highly tempered steel.

In my experience, approximately 80 per cent of these foreign bodies originate in the mushroomed edges of a cold chisel, a steel drill or a steel hammer. As a matter of fact, if mechanics would not use chisels or hammers with mushroomed edges, then the number of intraocular foreign bodies would be reduced at least 50 per cent.

Nature and Mechanics of Force Which Causes Penetration of Globe.—A blow to a piece of steel by a hammer, by iron or by a steel tool can cause a particle of metal to fly and penetrate the eyeball. However,

1. As far as I have been able to ascertain, my series represents by far the largest number of intraocular foreign bodies treated by one person in this or in any other country.

the contact of metal in machines rarely causes intraocular penetration of a foreign body.

In some of my cases, after removing a steel particle I have succeeded in getting the hammer and the chisel, i. e., the tools which the workman was using at the time of the accident. By examining them I have found the site where the metal chipped off and have been able to fit the particle removed from the eye into it. By this procedure the origin of the particle of steel was definitely proved.

With regard to the force which causes the particle of metal to fly from the mushroomed edge of a cold chisel or of the head of a hammer: A workman is striking the head of a chisel with a hammer. A certain blow, in all probability the so-called glancing blow, causes a small particle of the mushroomed edge to break off. The same blow causes the particle to rotate on its axis; that is to say, there is a small particle of metal with sharp edges traveling at a terrific speed and rotating on its own axis at the same time.

The effect is that of a flying buzz saw. As a matter of fact, if it were not for the buzz saw effect, i. e., if the particle were not rotating, it is questionable how many of the particles would penetrate the cornea and particularly the tough coat of the sclera, notwithstanding the fact that they travel at terrific speed.

Shape, Size and Location of the Bodies.—The shape and the size of the particles vary greatly. The most common shapes are oval, round oval and spear shaped. Almost all particles have one flat surface, one convex surface and extremely sharp edges. The size of the particles is from 0.5 to 6 or 8 mm. in diameter. In this connection, a particle measuring 0.5 mm. is difficult to show in a roentgenogram. In my series the wound of entrance was through the cornea in approximately 80 per cent of the cases.

My records show that 85 per cent of steel particles within the globe were located in the posterior chamber. The exact location might be in the vitreous, in the retina, in the choroid or in the sclera. Of the remaining 15 per cent the location was in the anterior chamber, in the lens, in the iris or posterior to the iris.

When not successful in removing what is undoubtedly a particle of steel from the interior of the eye, the ophthalmologist often believes that the particle is nonmagnetic. To be sure, there are certain steels with a high manganese content which are not as magnetic as ordinary steel. This type of steel cannot be called nonmagnetic; it can only be said that it is not as magnetic as ordinary steel.

It has been my experience that when a giant magnet fails to withdraw the steel, the particle is not nonmagnetic but is confined in an inflammatory exudate or embedded in the choroid or the sclera so deeply that the magnet is incapable of pulling it out.

DIAGNOSIS

Strange as it may seem, it is possible for a particle of metal to penetrate the globe and the injured person have little, if any, knowledge of an injury to the eye. In other words, it is possible for a small particle of metal to penetrate the conjunctiva and the sclera and lodge in the vitreous without the patient's being aware of any considerable pain or discomfort.

Obviously, such cases are extremely rare. Furthermore, among my large number of cases I have been able to determine the wound of entrance definitely in only 65 per cent.

Therefore, when a patient comes for treatment with a history of an injury to the eye capable of causing penetration of the eyeball, the first thought should be to exclude the possibility of an intraocular foreign body. The patient should be questioned as to what work he was performing when the eye first started to bother him.

If, for instance, he says that he was striking a chisel with a hammer, one should become suspicious of the presence of an intraocular foreign body. However, only roentgen examination will prove definitely the presence and the location of a foreign body.

On the other hand, if the patient says that he was grinding tools on an emery wheel, and one finds metal lodged on or embedded in the cornea, one does not think seriously of a metallic foreign body in the globe. I have never known a particle of metal to have penetrated the globe when a tool or steel was being ground on an emery wheel.

I would say that in all cases of acute inflammation of the eye occurring after the use of a hammer and chisel, notwithstanding the fact that there was no evidence of trauma within the eyeball, the eye should be examined by roentgenogram as a routine measure as a precaution against nondiscovery of an intraocular foreign body. As a matter of fact, when the ophthalmologist encounters an eye suffering from an inflammation of the middle or inner coats, that is, iritis, iridocyclitis, choroiditis or retinitis, particularly in a worker exposed to flying particles of steel, the possibility of the presence of an intraocular foreign body should be kept in mind until a roentgenogram has been made. It is, furthermore, to be borne in mind that persons not directly employed in the act of striking steel with a hammer sometimes suffer from this injury.

To go a step further, it is my belief that all eyes suffering from an inflammation whose presence cannot be accounted for by the usual diagnostic methods should be examined by roentgenogram in order to exclude the possibility of an intraocular foreign body.

Roentgenogram of the Eye.—Obviously, detection of a foreign body in the globe and its exact localization is exclusively the work of a radiologist who has made a specialty of this kind of work. I have found

in most instances that the work of an ordinary radiologist is worthless in determining the presence and the locality of an intraocular foreign body.

The most important part of a roentgen ray examination is to show the presence of a foreign body. The localization of the body is important principally to show whether or not it is within the globe. The localization is also important in that it may show, after the application of the magnet, whether or not the particle has moved.

Reaction of the Particle to the Magnet.—The reaction to the pull of the magnet is manifested, when the current is applied, either by a sudden pain or pulling sensation in the eye or by the appearance of the particle in the anterior chamber or posterior to the iris. The reaction of the foreign body to the magnet is of great importance as to prognosis. Absence of a reaction after the magnet has been applied intermittently for a period of five or ten minutes directly over the site of the foreign body might indicate:

1. That the foreign body, although localized by roentgenogram within the globe, is in reality outside the globe in the orbit.
2. That the particle is partially or entirely in the sclera.
3. That there is an inflammatory exudate covering the particle.
4. That the particle is only slightly magnetic.

It has been my experience that if there is no reaction on the first day the magnet is applied, a reaction is not likely to be obtained thereafter. However, notwithstanding an absence of reaction on the first day, one should apply the magnet for eight or nine succeeding days before deciding that it will not accomplish its purpose.

When there is a reaction, it is always possible to draw the foreign particle forward into the anterior chamber. Once it is lodged there, removal is easily accomplished.

In cases in which there is no reaction after the application of the magnet on successive days, another roentgenogram should be taken in order to determine whether or not the foreign body has been moved from its original location. If the roentgenogram shows that the particle has not moved and is within the globe or within the confines of the sclera, then the question of opening the sclera at the site of the foreign body must be considered.

Recently it has been asserted that particles of metal in the vitreous change their position owing to the force of gravity. In my series of cases none of the particles changed its position except when it was moved by the magnet.

It is true that a metallic body in the anterior chamber changes its position by gravity. The contents of the anterior chamber are fluid, and the structure of the vitreous is solid or semisolid; I cannot conceive,

therefore, how gravity would cause a particle to move in a normal vitreous.

Obviously, in a diseased and degenerated eye in which the vitreous was liquid a change in position might occur.

OPERATIVE PROCEDURE

When Particle Is in Anterior Chamber.—When a magnetic foreign particle is in the anterior chamber, in the iris, posterior to the iris or in the lens, if the wound of entrance is found in the cornea and has not healed it can be enlarged and the particle withdrawn through it. If the wound is located within the pupillary area, I prefer not to enlarge it. As a matter of fact, I have removed a particle through the wound of entrance in the cornea in very few cases. Otherwise the procedure is as follows:

1. Fully dilate the pupil and anesthetize the eyeball.

2. With the hand magnet pull the particle into the pupillary area and manipulate the magnet in such a way as to place the particle on the surface of the iris at a point midway between the upper margin of the cornea and the upper margin of the pupillary area. Then make a small keratome incision near the site of the particle and withdraw the particle through it.

When the foreign particle is in the anterior chamber, its removal is not difficult and the operation is invariably successful.

When Particle Is in Posterior Chamber.—In cases in which the foreign particle is in the posterior chamber, that is, in the vitreous or embedded in the coats of the eyeball, removal is more difficult. After a roentgenogram with correct localization of the particle has been made, the procedure is as follows:

After full dilation of the pupil, I at first disregard the locality of the foreign body and apply the giant magnet about 6 or 8 inches (15 to 20 cm.) from the center of the anterior surface of the cornea. The reason for applying the magnet at this distance from the cornea is to prevent unnecessary trauma to the iris or ciliary body caused by impact of the particle. If on application of the giant magnet at this distance there is no reaction, I gradually bring the magnet nearer to the surface of the cornea.

The reason for applying the magnet in the center of the cornea and not over the globe at the site of the foreign body is to draw the body around the lens into the pupillary area of the anterior chamber. In applying the magnet directly over the point of localization there is danger of pulling the particle beyond the periphery of the lens and having it become entangled in the iris. Full dilatation of the pupil prevents entanglement of the foreign body in the iris.

Anterior Versus Posterior Route.—In my series of cases I have found it necessary in only 6 to make an opening in the sclera and withdraw the particle through it. In these cases the particle was so firmly embedded in a tissue or exudate that the magnet could not dislodge it unless the tip came in close contact with it. As a matter of fact, I consider opening the sclera only as a last resort. I need not mention the dangers attendant on making an incision through all the coats of the eyeball sufficiently large to permit the entrance of the tip of a small magnet in case withdrawal at the site of the incision is not successful.

Many ophthalmologists advocate the removal of all intraocular foreign particles through the sclera, that is, by the so-called posterior route. The advocates of the posterior route contend that the eye is traumatized and particularly that in drawing the particle from the posterior chamber to the anterior chamber there is danger of an injury to the lens which will produce a cataract.

Admittedly, withdrawal by the anterior route can cause some trauma to the ciliary body or the iris, but I submit that the eye not only is traumatized to the same degree by withdrawal through the posterior route but in addition may be irreparably damaged by incision of the sclera. The contention that withdrawal via the anterior route may cause injury to the lens is entirely without substantiation. No such injury occurred in any of my cases.

Withdrawal of particles from the posterior to the anterior chamber does not injure the lens because:

1. In the comparatively slow movement of the particle the force of the impact is slight when it contacts the lens.

2. If the particle contacts the lens, the pull of the magnet draws it so that its flat surface and not the sharp cutting edge "slides" around the lens capsule.

Of all the dangers attendant on removal of a foreign particle by the posterior route, the greatest is the occurrence of detachment of the retina due to damage to the underlying coats of the eye attendant on the incision through the sclera.

The detachment may occur at any time after the operation, and many times it occurs after a period of months or even years. It is probably caused by contraction of scar tissue at the site of the operation. This causes traction on the retina, which is susceptible to detachment because of the fact that it only lies on the surface of the choroid and is held in place only at the optic disk and the periphery of the fundus at the ora serrata.

Removal by the anterior route has no disadvantages. Removal by the posterior route is more difficult and, according to published reports, not nearly as successful with regard to the operation itself and the after-

results. The injury to the eye by this procedure can be immeasurable. In view of my experience, it seems scarcely credible that any experienced ophthalmologist would undertake to remove a magnetic intraocular foreign body by making an opening through the sclera when the removal could be accomplished more easily and without danger of damage to the eye by way of the anterior chamber.

It is no wonder that ophthalmologists advocating the posterior route report extreme or even total loss of sight in a large percentage of their cases. The removal of magnetic particles by the posterior route as a routine measure should be discouraged.

RESULTS OF THE INJURIES

Before attempting a worth while appraisal of results one must consider:

1. Extent of damage to the eye by the injury itself.
2. Occurrence of infection.
3. Removability of the particle.
4. Function of the eye after removal of the particle.

In 2 per cent of my cases the initial injury caused irreparable loss of sight. Partial or total opacification of the lens occurred in 15 to 20 per cent of the cases. Serious infection before removal occurred in 4 cases. Removal of a particle which was presumably magnetic failed in 1 per cent of cases.

Excluding cases of irreparable damage by the initial injury, whether by injury to the lens or to other parts, and cases in which the particle could not be removed, one finds that in 80 to 90 per cent of the cases normal or slightly less than normal vision was retained after the removal.

SUMMARY AND CONCLUSIONS

It is a demonstrated fact that a large percentage of intraocular steel particles originate from the heads of steel hammers and the edges of chisels. It is obvious that if workmen did not use imperfect tools, injury with such particles would not occur.

The use of such tools could be prevented in three ways:

1. Education of workmen to be aware of the dangers attendant on using defective tools.

2. Daily inspection of hammers and chisels by the employer or the shop foreman to see that they are ground so that all overhanging edges are removed.[2]

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2. It is my opinion that if a person suffering from an injury due to the use of a defective tool such as I have described brought suit against his employer under the Employer's Liability Act the courts would find negligence on the part of the employer.

3. Insistence by the employer that the workmen who perform tasks in which there is danger of a foreign body's entering the eye wear non-breakable goggles or protective shields.

The eye of a patient giving a history of injury sustained while he was striking tempered steel with a hammer should be under suspicion until the presence of an intraocular foreign body is eliminated by roentgen ray examination.

The particle should always be removed through the anterior route except (1) in cases in which the wound of entrance is in the sclera and is sufficiently large for an attempt to be made to withdraw the foreign particle through it and (2) in cases in which the foreign body is confined in an inflammatory exudate or is entangled in either the choroid or the sclera and the magnet fails to pull it into the anterior chamber.

In all cases in which the particle is magnetic and is not embedded in the choroid or the sclera or confined in exudate, a giant magnet will draw it into the anterior chamber.

In the majority of cases in which the initial injury does not cause irreparable damage and no serious inflammation develops, the sight of the eye will not be seriously impaired.

Finally, I would emphasize that, although penetration of a particle of steel into the globe of the eye is the most serious of all injuries to the eye, if removal is done properly the results are infinitely better than previous observers have reported.

52 East Sixty-Eighth Street.

IRREGULAR AND MULTIPLE HOMONYMOUS VISUAL FIELD DEFECTS

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AND
I. S. WECHSLER, M.D.
NEW YORK

As is well known, the majority of lateral homonymous visual defects are of the hemianopic variety, while the quadrantanopic types are less often seen. In general the defects consist of a single large uniform scotoma extending over one or two quadrants on one side of the vertical meridian. They not infrequently spare the central field of vision and invariably implicate the peripheral parts.

Homonymous central or paracentral scotomas have been reported from time to time,[1] but multiple scotomas in homonymous fields are extremely rare. In reviewing the literature, Wilbrand and Saenger[2] cited several isolated instances of irregular and multiple field defects occurring in patients with gunshot wounds of the occipital lobe cortex and the adjacent optic radiations. In a large series of cases of tumors involving the geniculocalcarine pathways in which careful tangent screen and perimetric studies were made, isolated discrete scotomas were never found.[3]

The following is a report of 3 cases of multiple homonymous scotomas observed and studied over long periods at the Mount Sinai Hospital.

CASE 1.—A 39 year old Italian man was admitted to the hospital on Oct. 21, 1940 with severe headache, dizziness and blindness of ten hours' duration. In April 1940 he had an attack of dizziness, and in August of the same year he had another, during which objects moved around on a 45 degree angle to his right. He vomited on the two occasions.

On examination the patient was found to be poorly oriented, extremely restless and somewhat confused. His power of concentration and his memory for recent events were defective. He was unable to recognize objects. Except

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From the neurologic service of Dr. I. S. Wechsler, Mount Sinai Hospital.

1. Barkan, O., and Boyle, S. F.: Paracentral Homonymous Hemianopic Scotoma, Arch. Ophth. **14**:957-959 (Dec.) 1935. Newton, F. H.: Paracentral Homonymous Hemianopic Scotoma, Am. J. Ophth. **19**:600-601 (July) 1936.

2. Wilbrand, H., and Saenger, A.: Die Neurologie des Auges, Wiesbaden, J. F. Bergmann, 1917, vol. 7, pp. 259-313.

3. Bender, M. B., and Kanzer, M. G.: Dynamics of Homonymous Hemianopias and Preservation of Central Vision, Brain **62**:404-421 (Oct.) 1940.

for perception of movement in the right lower quadrants of his fields of vision, he was practically blind. He had left spastic hemiparesis of the faciobrachial type, a Chaddock sign on the left and impaired sensation in the arm and the face. The optic fundi and the pupillary reactions were normal.

The white blood cell count on admission was 17,000 per cubic millimeter, with a normal differential count. The Wassermann reaction of the blood and of the spinal fluid were negative. The spinal fluid was under an initial pressure of 90 mm. of water and contained a total protein content of 38 mg. per hundred cubic centimeters and no cells.

Under observation, the patient complained of severe headache. For a few days he had a low grade fever, with a temperature up to 100.6 F. He was disorientated in all spheres, and he kept calling for his mother. He complained that he was unable to judge distances and that his vision was bad. Perimetry could not be performed because of his mental state. On October 25 his sight

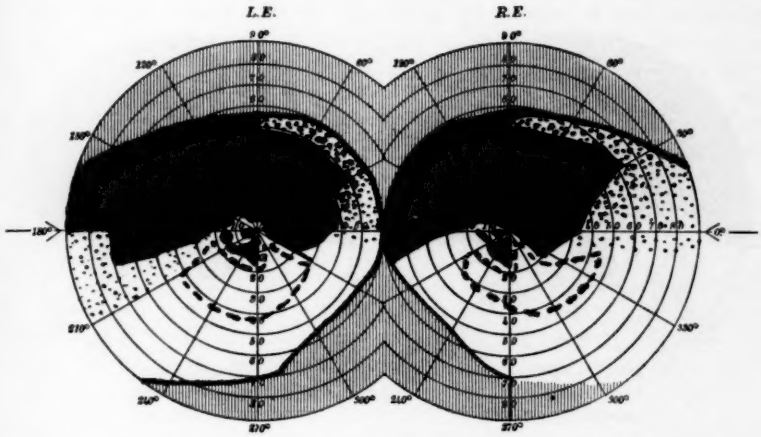

Fig. 1 (case 1).—Perimetric fields with daylight illumination and test objects varying in size from 2 to 10 mm. The smaller the object, the larger the scotoma. The solid black areas represent zones of absolute blindness. The stippled areas are those in which object movement was recognized but not form or color. The areas of preserved color vision are indicated by broken lines. Note that all four quadrants were implicated.

began to improve. He was able to identify a pencil, a combination fountain pen, a flashlight and a safety pin, but not coins or colors. Visual field examinations were unsuccessful chiefly because the acuity varied from time to time at any given points. When he was asked to read whatever letters he saw in a group placed before him he invariably chose those in the right lower quadrants.

In addition to the visual difficulties, he complained bitterly of severe itching and burning sensations in the left hand, although objectively the signs of motor and sensory defects, including dysterognosis, had disappeared. He also had spells of involuntary crying. He continued to have trouble in finding objects around him. He was unable to feed himself because of the visual difficulties.

A roentgenogram of the skull showed nothing abnormal. A pneumoencephalogram and an electroencephalogram were essentially normal. The

plotted visual fields showed defects in all four quadrants but chiefly in both upper quadrants; scotometry could not be performed with any reliability until a later date (figs. 1 and 2). On November 1 the patient showed some improvement in his psychic and visual status. He regained some insight and orientation. Until then he had not realized that he was ill, and he had thought he was in another hospital. Apparently he had total amnesia for the first eleven days in the hospital. He still had no conception of time. For many days he had spells of involuntary crying, but between them he seemed excessively cheerful. He showed marked disturbance in spatial orientation. He was allowed to walk about the ward, but he was unable to find his way to his bed. He could not remember direction as to right or left and could not retrace his steps. His surroundings seemed unfamiliar to him. He could not remember objects which he saw. He was discharged on November 13 and referred to the follow-up clinic.

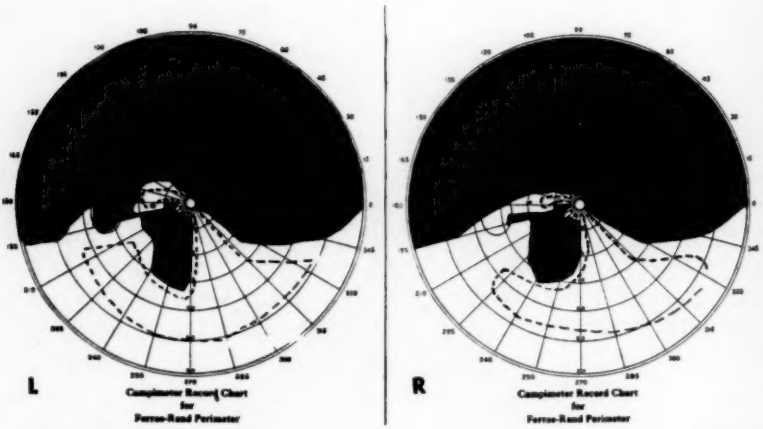

Fig. 2 (case 1).—The central fields by campimetry with daylight illumination and test objects varying in size from 2 to 10 mm. The solid black areas represent zones of absolute blindness. The areas of preserved color vision are indicated by broken lines. Homonymous scotomas are depicted in the left inferior quadrants. Note that the macular regions for 2 degrees are spared and that color vision is preserved in these areas.

When he left the hospital, he complained that everything seemed strange to him. He confused streets and their directions. He felt insecure in his travels because he could not tell which was north, east, south or west. He feared that he might lose his way, chiefly because the streets seemed unfamiliar. He also had trouble in recognizing simple objects, such as a key. His visual memory was grossly defective. When he looked at something in a window he could not think of what he had seen until a few minutes had passed. Once he searched for a comb which he had seen and knew was on the bureau, but he could not find it for a short while. He complained that his memory was defective; he could not recollect where he placed objects. Although he was able to read he could not recall what he saw until two or three days later. He had visual hallucinations and other psychovisual disturbances, which are best expressed by

his productions. In an interview in the follow-up clinic on Aug. 12, 1941 he made the following statements:

"Sometimes I looked for something which I could not find, and this made me nervous. I was unable to figure as quickly as before; my mind could not record the numbers quickly. I was unable to divide or multiply quickly, and this made me nervous, and my head would swell up.

"I think there was something wrong with my recording sense. The things you see or write are supposed to record, but with me it takes a long time to make an impression on me. Everything seems slow. I can't think so fast. I don't remember everything I see, and therefore I can't work. The little things I forget for a short while; then it all comes back suddenly without any mistakes and with a feeling of well-being. I still have trouble with my sense of direction as to left and right. Time is not fast or slow—it is normal. When I got out of the hospital, time went very fast—day and night.

"Everything appears almost normal now, but when I got out of the hospital, everything seemed strange. On getting out of the hospital I was on Madison Avenue but I thought I was on Fifth Avenue. Also I thought the taxi was going downtown but it really was going uptown. During the day I see images of rising water which spreads out slowly from the center. The images move around and seem to be slanting in front of me. On looking steadily at one spot I see half of objects on my left and then a quarter, and then the objects are covered by the image of rising water.

"During the night I see white pointy spots and lightning streaks in front of me. Some are slanty and ripple along. I cannot distinguish dark colors, especially blue and black."

The visual fields have remained the same as those plotted in figure 1. The spatial disorientation, optic agnosia and defective visual memory have disappeared. The patient still has hallucinations of small scintillating lights in his upper right quadrants. There is no optic nerve atrophy, nor are there disturbances in the pupillary light reflex, their absence indicating that there is no chiasmal lesion.

There is no doubt that the patient had involvement of both occipital lobes. This assumption is supported not only by the visual defects in all four quadrants but by the remarkable psychovisual disturbances, which are usually due to disease of the lateral portion of the occipital lobe cortex.[4] Fleischer[5] described similar homonymous defects in all four quadrants in a patient with a gunshot wound of the occipital lobe. The transient sensory and motor defects found on the left side of the body and the involuntary weeping indicate that the lesion was more extensive and involved both sides of the brain. Since there were a low grade fever and slight leukocytosis, the probability is that the process was inflammatory.

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4. (*a*) Brain, W. R.: Visual Object Agnosia with Special References to the Gestalt Theory, Brain **64**:43-62 (March) 1941. (*b*) Holden, W. A.: A Case of Mind Blindness, Unique in That the Entire Mesial Surface of Both Occipital Lobes and Both Optic Radiations Were Preserved, Tr. Am. Ophth. Soc. **10**:286-290, 1904.

5. Fleischer, cited by Wilbrand and Saenger.[2]

The unusual feature in the visual fields in this case is the multiple defects implicating all quadrants. Judging from the fields, the greatest damage occurred in the inferior portion of the calcarine cortex, particularly on the right, resulting chiefly in an altitudinal hemianopia. Altitudinal hemianopia is usually due to lesions of the occipital lobes.[6]

CASE 2.—A 42 year old bartender entered the hospital on Jan. 22, 1940 because of a sudden attack of dizziness and blindness. Four years before admission he had an attack of coronary occlusion, from which he recovered. Seventeen days before admission, on kneeling to pick up a bottle, he suddenly became dizzy and had a sensation of reeling to the right. When he raised himself, he found he was totally blind. In the course of the next two days his vision began to return, so that he could see objects in the lower left quadrants. During the past week, he had imagined that he saw small men on each side of him. This hallucination

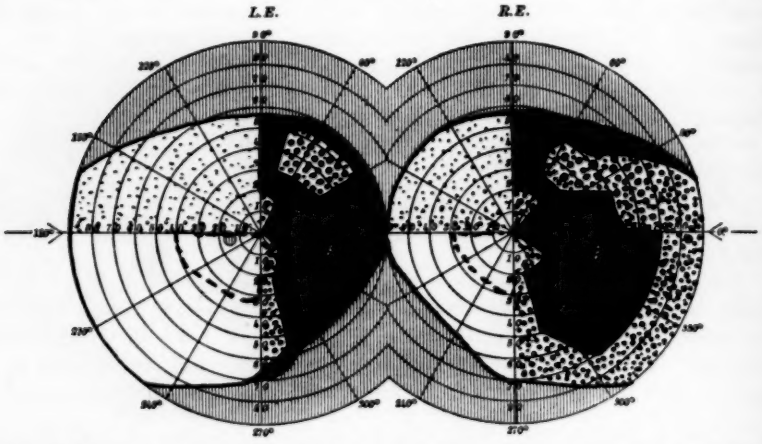

Fig. 3 (case 2).—Perimetric fields with daylight illumination and test objects varying in size from 2 to 10 mm. The only areas normal for form and for color vision are in the left inferior quadrants. In the left upper quadrants (light dots) the patient recognized movement but not color or form. There is also incomplete right homonymous hemianopia (solid black) with islands of relative vision in the central, paracentral and peripheral fields, represented by heavy and light dots. Note the sector defects in the central fields and the temporal crescents in the peripheral fields, both characteristic of lesions of the occipital lobe.

recurred on several occasions and usually at night. On one occasion he thought he saw a "normal-sized man."

The physical examination revealed signs of aortic and mitral stenosis. The blood pressure was 106 systolic and 74 diastolic. The neurologic examination gave negative results. The visual fields disclosed homonymous field defects, as plotted in figure 3. The optic papillae and the pupillary reactions were normal.

6. Uhthoff, W.: Beiträge zu den hemianopischen Gesichtsfeldstorüngen nach Schädelschussen, besonders solchen im Bereich des Hinterhauptes, Klin. Monatsbl. f. Augenh. **55**:104-125, 1915.

The Wassermann reaction of the blood and of the spinal fluid was negative. The spinal fluid was under a pressure of 220 mm. of water and contained a total protein content of 47 mg. per hundred cubic centimeters and no cells. A roentgenogram of the skull was normal. An electroencephalogram showed depression of the alpha activity in the left occipital lobe.

During the patient's first week in the hospital, he frequently had visual hallucinations. He imagined that he saw several people from the corner of his right eye, but when he turned his head to the left the images disappeared. The images were usually of people whom the patient knew. Each hallucination was of a single person who appeared to be approaching from the right and disappeared just in front of the patient or when he looked toward the right. The images were colorless, were unaccompanied by auditory hallucinations, did not appear to be engaged in any special activity and did not arouse anxiety. He did not believe they were real and recognized them as visual disturbances. In the beginning they seemed to be diminutive figures, but later they turned out to be the size of normal human beings. He remarked that since his vision had improved the images no longer appeared.

Since the patient's cardiac status was poor, a pneumoencephalogram was not made. He was discharged from the hospital on February 6 and observed in the follow-up clinic. When last examined, in January 1942, he showed no change in his status. The visual fields were unchanged, and he had no new complaints. The optic papillae and the pupillary reactions were normal.

This case is an example of double lateral hemianopia with islands of spared vision. It is not likely that the bilateral visual field defects were due to a chiasmal lesion, since there was no optic nerve atrophy or disturbance in pupillary function. The field defects must have been due to a lesion somewhere in the geniculocalcarine pathway. The tendency to spare the "temporal crescent" in the right field of vision and the sector-like defect in the central fields suggest that the occipital lobe was involved.[7]

Apparently the damage was bilateral and because the patient had cardiac disease and the onset was sudden the clinical picture may be explained by vascular lesions in both occipital lobes.

CASE 3.—A 45 year old Italian ice dealer entered the hospital on Feb. 15, 1938 because of impairment of vision. Two weeks before admission he suddenly noted that he was unable to see in the right field of vision, and when he closed his left eye he could not see at all unless he shifted his head so as to fixate an object. He also noted that on shifting his gaze to the right at a bright object directly

7. Bender, M. B., and Strauss, I.: Defects in Visual Field of One Eye Only in Patients with a Lesion of One Optic Radiation, Arch. Ophth. **17**:765-787 (May) 1937. Delépine, S.: Case of Right Lateral Homonymous Hemianopsia, Arteriosclerotic Softening of the Left Cuneus, Tr. Path. Soc. London **41**:2-16, 1890. Behr, C.: Die homonymen Hemianopsien mit einseitigem Gesichtsfelddefekt, im "rein temporalen halbmondförmigen Bezirk des binokularen Gesichtsfeldes," Klin. Monatsbl. f. Augenh. **56**:161-172, 1916. Uhthoff.[6]

before him, he saw flashes of light of different colors, especially red, blue and green, in his right field of vision. He also complained of dizziness on walking, as if he were drunk, with a tendency to reel to the right. He stated that objects and his immediate surroundings seemed to rotate in a horizontal plane around his vertical axis.

General examination revealed no gross abnormalities. The blood pressure was 152 systolic and 95 diastolic. The neurologic status was normal. The visual fields revealed several irregular homonymous multiple scotomas in the right field of vision (fig. 4). These were not discovered until several perimetric examinations were made. A lumbar puncture showed clear and colorless fluid which was under a pressure of 110 mm. and contained a total protein content of 58 mg. per hundred cubic centimeters and 4 lymphocytes.

Under observation the patient complained of buzzing in the right ear and objective clockwise vertigo. Caloric tests gave normal results. A roentgenogram of the skull was normal, and so was an electroencephalogram.

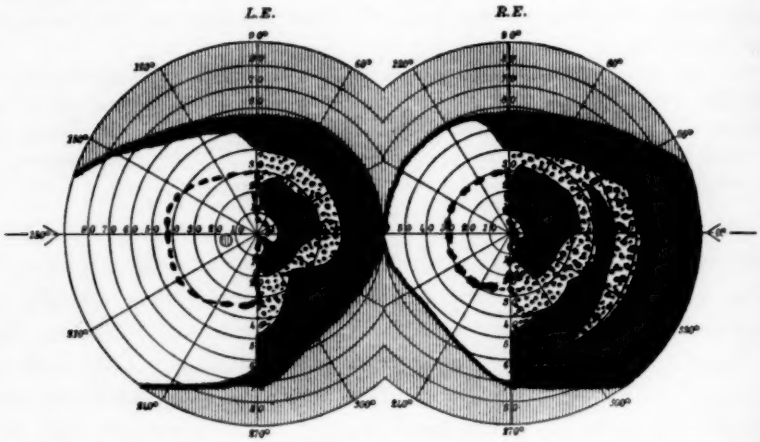

Fig. 4 (case 3).—Perimetric fields with daylight illumination and test objects varying in size from 4 to 10 mm. There is right homonymous hemianopia (solid black) with islands of relative vision (dotted areas) in the paracentral and central fields. Note the large paracentral scotomas.

The patient was discharged on March 8 to the follow-up clinic, where he has been observed for over four years. Except for a "mild cerebral accident" for which he was treated at another hospital in 1941, there were no new symptoms. His visual field defects have remained unchanged (January 1942).

The slightly elevated blood pressure and the subsequent history of a "cerebral accident" indicate that the most likely explanation for the visual disturbance is a vascular lesion in the geniculocalcarine pathway. The lack of progression obviously excludes tumor. The localization is difficult. Lateral homonymous multiple scotomas have rarely been found in association with lesions of the optic radiations, whereas they have been observed in association with lesions of the occipital lobe. Similar

visual field defects have been described by Wilbrand [8] in a case in which postmortem examination disclosed arteriosclerotic softening of the occipital lobe cortex.

COMMENT

In the absence of definite knowledge of the pathologic processes and their localization, it is difficult to correlate accurately the clinical findings with anatomic structures. However, there are a number of clinical facts which justify an expression of opinion as to the probable location of the lesion. Furthermore, the peculiar nature of the special field defects shown by the patients fortifies in some measure the deductions which we wish to make.

Field defects obviously can occur as a result of a lesion anywhere in the course of the visual pathways, from the retina through the optic nerve, chiasma, tract, geniculate ganglion and optic radiation to the occipital cortex. The absence of retinal changes and optic nerve atrophy at once excludes a lesion in the optic pathway to the geniculate bodies. The preservation of the light reflex and the absence of optic nerve atrophy eliminate a chiasmal lesion. Acute bilateral hemianopic field defects due to a chiasma lesion are usually incongruent, involve chiefly the temporal quadrants and are rarely, if ever, homonymous. The homonymous hemianopias in our cases were due to lesions either in the optic radiation or in the calcarine cortex. While one cannot positively say that the lesion is not in the optic radiation, a number of the clinical facts point to the cortex as the more probable localization.

In the first place, bilateral lesions are not likely suddenly to affect the radiation, while such lesions are known to occur in the cortex. Secondly, because of the compactness of the fibers in the radiation, a lesion there is apt to result in a homogeneous field defect. On the other hand, bilateral homonymous hemianopia can and does occur as a result of a lesion in the occipital cortex. Sufficient recovery may rapidly ensue in the case of a cortical lesion, and the wide area covered by the visual cortex permits of irregular involvement and the occurrence of numerous scotomas in the homonymous fields. Finally, there is the additional evidence, at least in the first 2 cases, of cortical involvement other than the occipital. The visual hallucinations and disturbances in spatial orientation obviously reflected lesions of the cortex and not of fiber structures.

There remains the question whether the lesion is in the lateral occipital cortex or in the calcarine area. In case 1 the object agnosia, defective visual memory and disturbance of spatial orientation point to the outer surface of the occipital lobe, but the visual field defects speak

8. Wilbrand, H.: Die hemianopische Gesichtsfeldformen, Wiesbaden, J. F. Bergmann, 1890, pp. 54-60.

for the calcarine cortex or the terminal portion of the optic radiation.[4b] This is particularly true of the upper field defects, altitudinal hemianopia, which generally reflect bilateral involvement of the lower lips of the calcarine cortex. It may well be that in the case in which there was spatial disorientation, the external surface was also involved, either by an extensive lesion or by edema, which receded sufficiently to bring about partial recovery and left behind only the multiple scotomas in the homonymous fields due to the residual lesion in the striate cortex.

SUMMARY

Three cases of irregular and multiple homonymous field defects are described. The bilaterality and the nature of the scotomas and the associated visual symptoms suggest that the lesion in each case was in the calcarine cortex of the occipital lobe.

Fifth Avenue and One Hundredth Street.

Ophthalmologic Reviews

EDITED BY DR. FRANCIS HEED ADLER

RECENT CONTRIBUTIONS TO LOCALIZATION OF VISION IN THE CENTRAL NERVOUS SYSTEM

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BALTIMORE

The occipital lobe of mammals is divided into three regions, each distinguished by a peculiar cytoarchitecture. In primates, the region which surrounds the calcarine fissure, the area striata, is characterized by an unusually broad band of medullated fibers, visible to the unaided eye, the stripe of Gennari.[1] This stripe passes through the fourth cellular layer of the primary cortical center for sight. Surrounding the area striata lies the area parastriata (area 18 of Brodmann,[2] OB of von Economo and Koskinas[3]), and around the area parastriata, the area peristriata (area 19 of Brodmann, OA of von Economo and Koskinas). The total depth of the cellular layers of these three divisions of the occipital lobe varies slightly, as follows: 2.41 mm. for area 17, 2.32 mm. for area 18 and 2.46 mm. for area 19. In Bolton's[4] terminology, the granular layer, i. e. the fourth layer, makes up about one third (0.82 mm.) of the total depth of area 17, one thirteenth in area 18 (0.18 mm.) and one tenth in area 19 (0.24 mm.). The infragranular layers, i. e. the fifth and the sixth layer, make up, respectively, 40, 35 and 38 per cent of the total depth for each division from within outward, i. e. 0.95 mm., 0.86 mm., and 0.92 mm., while the supragranular layers, i. e. the first to the third, form less than one third of the total depth of area 17 (0.64 mm.) and more than one half of the total depth in both area 18 and area 19 (i. e. 1.28 and 1.30 mm.) respectively. The receptive layer (sixth) of area 17 is divided into three layers, one above and one below the stripe of Gennari; but the supragranular layers, particularly the

Lecture given at the Harvard Medical School on March 17, 1942 under the auspices of the Howe Laboratory of Ophthalmology.

1. Gennari, F.: De peculiari structura cerebri, nonnullisque ejus morbis, Parma, ex regio typog., 1782.

2. Brodmann, K.: Vergleichende Lokalisationslehre der Grosshirnrinde in ihren Prinzipien dargestellt auf Grund des Zellenbaues, Leipzig, J. A. Barth, 1909.

3. von Economo, C., and Koskinas, G. N.: Cytoarchitektonik der Hirnrinde des erwachsenen Menschen, Vienna, Julius Springer, 1925.

4. Bolton, J. S.: The Brain in Health and Disease, London, J. Arnold, 1914.

third, show further subdivision in both area 18 and area 19—a characteristic of areas in which interregional connections are important.

Certain cellular characteristics of these three divisions of the occipital lobe are noteworthy. The large solitary cells of Meynert in the fifth layer (Vb) of the area striata suggest that the primary receptive center

Fig. 1.—Section (× 5) through the medial surface of the occipital lobe of a human being, showing the distribution of axis cylinders in the three areas. Area 17 of Brodmann, the area striata (OC of Economo and Koskinas), surrounds the calcarine fissure (*f. c.*). Area 18 of Brodmann (OB of Economo and Koskinas), the area parastriata, surrounds the area striata, and area 19 of Brodmann, the area peristriata, surrounds the area parastriata.

itself projects on some motor area. Giant pyramids characterize the fifth layer of that part of the area peristriata (OBy) which surrounds the area striata and the third and fifth layers of that part of the area

parastriata (OA_2) which bounds the occipital lobe. The cytoarchitecture of these divisions of the occipital lobe suggests that the receptive center for sight in man is surrounded successively by an optic motor area and by an optic association center in which some motor activity must be present. Some part of each of the three regions appears to be cytoarchitecturally differentiated as an origin of corticifugal systems.

Before 1915 confusion characterized the localization of the primary cortical center for sight itself. On the one hand stood von Monakow,[5] supported in part by Winkler,[6] and on the other, Henschen,[7] with Mott,[8]

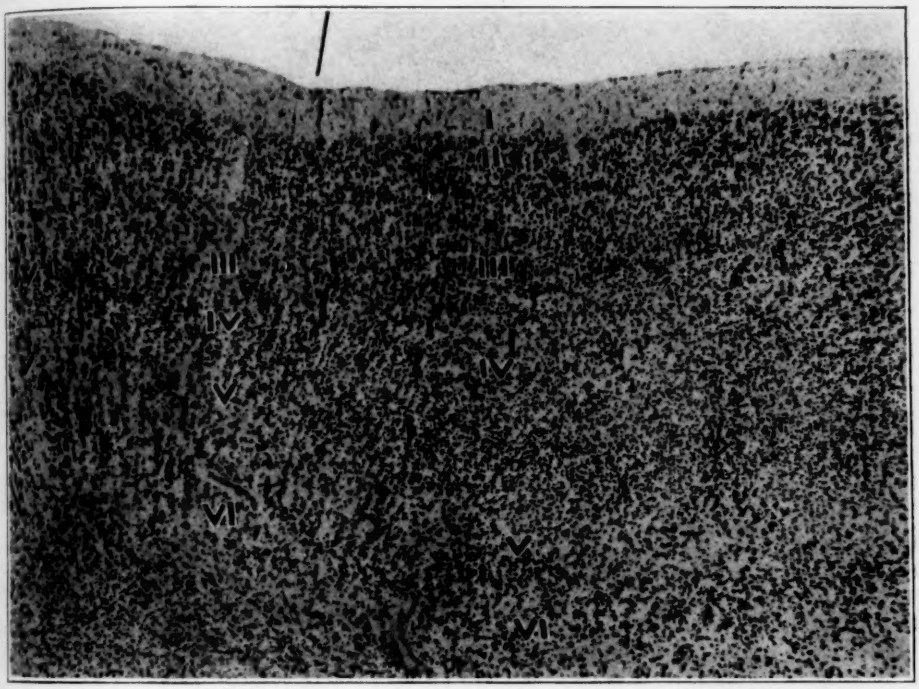

Fig. 2.—Section (× 40) showing the sharp demarcation between the area striata (OC) and the area parastriata (OB). Note the breadth of layer *IV* in the former region and the large pyramidal cells in the lower part of layer *III* in this division of the latter region. (From von Economo and Koskinas.[3])

5. von Monakow, C.: Gehirnpathologie, Vienna, A. Hölder, 1905.

6. Winkler, C.: Manuel de neurologie, Haarlem, E. F. Bohn, 1927, vol. 1, pp. 86-121.

7. Henschen, S. E.: Vierzigjähriger Kampf um das Sehzentrum und seine Bedeutung für die Hirnforschung, Ztschr. f. d. ges. Neurol. u. Psychiat. **87**:505-535, 1923.

8. Mott, F. W.: The Progressive Evolution of the Structure and Functions of the Visual Cortex in Mammalia, Arch. Neurol. Path. Lab. Lond. County Asyl. Claybury **3**:1-48, 1907.

Wilbrand,[9] Lenz[10] and Inouye,[11] the former postulating a diffuse cortical projection of secondary optic fibers covering at least the whole occipital lobe, and the latter, a geniculocortical projection confined solely to the area striata. After 1915 the publication of the results of a comparison of losses in visual fields with circumscribed traumatic lesions of the occipital lobe (Marie and Chatelin,[12] Holmes and Lister,[13] Holmes[14]) substantiated Henschen's quadrant projection of the retina on the area striata. For the first time von Monakow failed to challenge such results. Henschen's generalization stood.

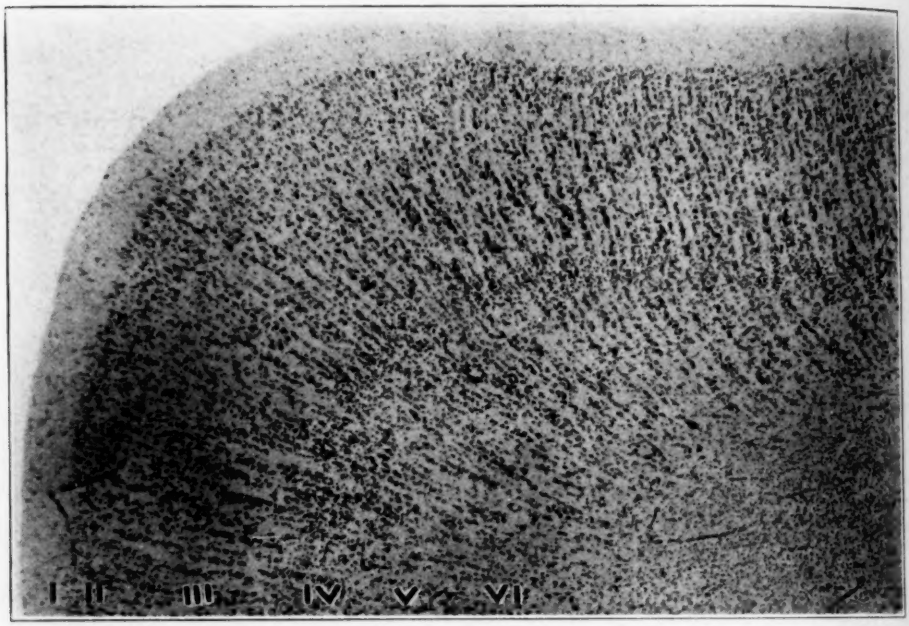

Fig. 3.—Section (× 25) showing the cytoarchitecture of the posterior division of the area peristriata (OA). All six layers are well laminated. Layer IV is broader than it is in the area parastriata (fig. 2) and narrower than it is in the area striata (fig. 2). (From von Economo and Koskinas.[3])

9. Wilbrand, H.: Ueber die makulär-hemianopische Lesestörung und die von Monakowsche Projektion der Makula auf die Sehsphäre, Klin. Monatsbl. f. Augenh. **45**:1-39, 1907.

10. Lenz, G.: Zur Pathologie der cerebralen Sehbahn unter besonderer Berücksichtigung ihrer Ergebnisse für die Anatomie und Physiologie, Arch f. Ophth. **72**: :1-85 and 197-273, 1909; Die Kriegsverletzungen der zerebralen Sehbahn, in Lewandowsky, M.: Handbuch der Neurologie, Berlin, Julius Springer, 1924, vol. 1, pp. 668-729.

11. Inouye, T.: Die Sehstörungen bei Schussverletzung der korticalen Sehsphäre, Leipzig, 1909.

Holmes [15] outlined this retinal projection on the area striata in a clearcut manner. The line of separation between the temporal and the nasal half of the retina was projected ipsilaterally respectively on the dorsal and on the ventral lip of the calcarine fissure and the horizontal line between the dorsal and the ventral quadrants in the depth of this fissure. The macula lutea was projected posteriorly, the peripheral margin anteriorly and the regions between in a series of arcs between the lip and the depth of the fissure and between the tip and the root of the fissure. The macula thus was not given bilateral representation. This minute localization of retinal fields within the area striata was generally accepted. The acceptance produced two immediate results. It almost silenced expression of the bilateral representation of macular vision, and it stimulated experimental research on the termination of the optic tract within the primary optic centers of the midbrain and thalamus. For it is obvious that localization of retinal fields on a cortical surface necessitates localization within the primary thalamic center itself.

That such a localization within the lateral geniculate body exists was suggested by case reports of Rönne [16] and Henschen.[17] Rönne found that only the cells in the dorsal part of the lateral geniculate body atrophied subsequent to degeneration of the macular bundle in cases of chronic intoxication involving the optic nerve. Henschen described a lesion of the medial part of the lateral geniculate body accompanied by a ventral quadrant hemianopsia. A complete exploration of the areas of retinal projection on primary optic centers is possible only in animals. Lubsen [18] was the first investigator not only to attempt such

12. Marie, P., and Chatelin, C.: Les troubles visuels dûs aux lésions des voies optiques intra-cérébrales et de la sphère visuelle corticale dans les blessures du crâne par coup de feu, Rev. neurol. **28**:882-925, 1915,

13. Holmes, G., and Lister, W. T.: Disturbances of Vision from Cerebral Lesions, with Special Reference to the Cortical Representation of the Macula, Brain **39**:34-73, 1916.

14. Holmes, G.: (*a*) Disturbances of Vision by Cerebral Lesions, Brit. J. Ophth. **2**:353-384, 449-468 and 506-516, 1918; (*b*) The Cortical Localization of Vision, Brit. M. J. **2**:193-199, 1919.

15. Holmes, G.: (*a*) A Contribution to the Cortical Representation of Vision, Brain **54**:470-479, 1931; (*b*) The Representation of the Mesial Sectors of the Retinae in the Calcarine Cortex, Jahrb. f. Psychiat. u. Neurol. **51**:39-48, 1934; (*c*) footnote 14 *b*.

16. Rönne, H.: Die anatomische Projektion der Macula im Corpus geniculatum externum, Ztschr. f. d. ges. Neurol. u. Psychiat. **22**:469-485, 1914.

17. Henschen, S. E.: Ueber Localisation innerhalb des äusseren Knieganglions, Neurol. Centralbl. **17**:194-199, 1898.

18. Lubsen, J.: Projection of the Retina on the Tectum Opticum in Leusiscus Rutilus, Nederl. tijdschr. v. geneesk. **67**:1258, 1923.

an experiment but to utilize the skill of an ophthalmologist. The animal chosen was a fish, Leusiscus rutilus, and the ophthalmologist, a professor at the University of Amsterdam, W. P. C. Zeeman. The results of that experiment were twofold: (1) the retinal quadrants were localized within the opposite tectum, and (2) an ophthalmologist was shown to be invaluable as a collaborator for a neurologist.

PRIMARY OPTIC CENTERS

The primary optic centers of vertebrates are located (1) within the tegmentum of the midbrain and thalamus, (2) within the roof of the midbrain and (3) within the dorsolateral division of the thalamus. In some mammals the optic tract penetrates the subthalamus, the generalized midbrain tegmentum, the substantia nigra, the oculomotor nucleus and the basal nucleus of the optic tract via two specialized divisions of that tract known, respectively, as the anterior and the posterior accessory optic tract. In all vertebrates, without exception, the optic tract enters the optic tectum, or the superior colliculus. In animals in which the thalamic centers for sight either are not present or are immature, the optic tectum serves as a primary receptive area and a region of higher correlation. In some mammals this part of the optic tract also terminates within the pretectal area.

The dorsolateral division of the thalamus contains two nuclei which have been described as primary centers for sight, the lateral geniculate body and the pulvinar. The lateral geniculate body is generally recognized as consisting of two easily delimited parts, the dorsal nucleus and the ventral nucleus. The optic tract terminates in the ventral nucleus in some mammals and in the dorsal nucleus in all mammals. The status of the pulvinar as a primary optic center has been definitely challenged for many mammals.

Primary Tegmental Centers.—The distribution of fibers of the optic tract to the tegmentum of the midbrain and thalamus has been studied recently by Gillilan [19] in six different orders of mammals. She stated the belief that the anterior accessory optic tract is related to nocturnal vision, for in animals with such vision well developed the nerve fibers of this accessory tract were large and heavily myelinated. The anterior accessory tract, which projects directly into the subthalamus, was identified in chiropters, insectivores and rodents but not in carnivores or in primates. The posterior accessory tract, which ends in the nuclei and tegmentum of the midbrain, was identified as a medullated tract in ungulates and in the three orders in which the anterior tract was found

19. Gillilan, L. A.: The Connections of the Basal Optic Root (Posterior Accessory Optic Tract) and Its Nucleus in Various Mammals, J. Comp. Neurol. **74**:367-408, 1941.

and as an unmedullated tract in the cat and in the monkey. Packer [20] found both of these tracts in a phalanger; Bodian,[21] only the posterior accessory tract in the opossum, and Jefferson,[22] neither of them in the ferret. In adult man no one has seen the posterior accessory tract since 1903 (Marburg [23]).

The Optic Tectum.—The optic tectum, or the superior colliculus, of mammals, including man, is made up of alternating fiber and cell layers, four of each. Of these layers, the superficial, or outer fiber layer, and the second fiber layer, the stratum opticum, come within the scope of the present review. It has already been noted that the retinal quadrants of Lubsen's fish formed a pattern within the opposite tectum. Following Lubsen, Ströer [24] reexamined the optic tracts of a series of representative vertebrates and found that the optic nerve left the retina in two main groups of fibers separated by a line which joined its two embryonic plicas, the inferior and the superior (Rabl [25]). This dividing line separated the posterior and ventral quadrants from the dorsal and anterior quadrants of the primitive vertebrate eye. Further, the two bundles remained separate within the optic tract and terminated separately within the optic tectum, the posterior-ventral division, anterior to the dorsal-anterior division. The two quadrants were further separated by dorsal and ventral terminations within the anterior or the posterior half of the tectum. This neat arrangement, which springs from a common vertebrate type of development of the optic cup, lies at the basis of the nasotemporal symmetry of higher animals (Rabl [25]).

Stimulated by the work of Ströer, Herrick [26] reexamined the visual systems in three lower forms. In each he found the optic nerve on leaving the retina divided into four divisions arising from four quadrants.

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20. Packer, A. D.: An Experimental Investigation of the Visual System in the Phalanger, Trichosurus Vulpecula, J. Anat. **75**:309-329, 1941.

21. Bodian, D.: An Experimental Study of the Optic Tracts and Retinal Projection of the Virginia Opossum, J. Comp. Neurol. **66**:133-144, 1937.

22. Jefferson, J. M.: A Study of the Subcortical Connexions of the Optic Tract System of the Ferret with Special Reference to Gonadal Activation by Retinal Stimulation, J. Anat. **75**:106-134, 1940.

23. Marburg, O.: Basale Opticus Wurzel und Tractus peduncularis transversus, Arb. a. d. neurol. Inst. a. d. Wien. Univ. **10**:66-80, 1903.

24. Ströer, W. F. H.: Zur vergleichenden Anatomie des primären optischen Systems bei Wirbeltieren, Ztschr. f. Anat. u. Entwcklngsgesch. **110**:301-321, 1939.

25. Rabl, C.: Ueber die bilaterale oder nasotemporale Symmetrie des Wirbeltierauges, Arch. f. mikr. Anat. **90**:261-444, 1918.

26. Herrick, C. J.: Development of the Optic Nerves in Amblystoma, J. Comp. Neurol. **74**:473-534, 1941; Optic and Postoptic Systems of Fibers in the Brain of Necturus, ibid. **75**:487-544, 1941; The Eyes and Optic Paths of the Catfish, Ameirus, ibid. **75**:255-286, 1941.

In the catfish and in Necturus, quadrant projection was not present in the optic tectum. In Amblystoma, the large axons springing from the retina developed during the larval stages and penetrated the superficial layer of the midbrain roof, the tegmentum and the hypothalamus at random; but about the time of metamorphosis the small axons within the optic nerve increased in number and on entering the optic tectum formed a retinal mosaic. Apparently, then, the projection of a retinal pattern on the midbrain, although phylogenetically old, is not present in all lower forms.

Brouwer and his collaborators [27] described a clearcut quadrant projection of the retina within the contralateral superior colliculus of the rabbit but failed to find a similar one in the cat and in the monkey. In the cat's tectum all the degenerated optic fibers were crossed fibers; in the monkey's tectum the majority were crossed and no macular fibers were present. Bodian [21] found that the projection of retinal quadrants on the superior colliculus of the opossum agreed essentially with that given by these workers for the rabbit as well as with that published by Lashley [28] for the rat.

The nerve fibers which make up the retinal mosaic in the tectum of the rabbit are small fibers (Bishop and O'Leary [29]), for the large fibers terminate in the lateral geniculate body. Primary optic fibers of small caliber and small-fibered collaterals (from large fibers destined for the lateral geniculate body) penetrate the stratum opticum of the contralateral superior colliculus in the cat (Barris, Ingram and Ranson [30]). So far no one has correlated the size of nerve fibers within the optic nerve and tract of the monkey with their central distribution.

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27. Brouwer, B.; Zeeman, W. P. C., and Houwer, A. W. M.: Experimentell-anatomische Untersuchungen über die Projektion der Retina auf die primären Opticuszentren, Schweiz. Arch. f. Neurol. u. Psychiat. **13**:118-138, 1923. Brouwer, B., and Zeeman, W. P. C.: Experimental Anatomical Investigations Concerning the Projection of the Retina on the Primary Optic Centers in Apes, J. Neurol. & Psychopath. **6**:1-10, 1925; The Projection of the Retina in the Primary Optic Neuron in Monkeys, Brain **44**:1-35, 1926. Brouwer, B.: Anatomical, Phylogenetical and Clinical Studies on the Central Nervous System, Baltimore, Williams & Wilkins Company, 1927. Overbosch, J. F. A.: Experimenteel-anatomische onderzoekingen over de projectie der retina in het centrale zenuwstelsel, Thesis, Amsterdam, H. J. Paris, 1927.

28. Lashley, K. S.: The Mechanism of Vision: VII. The Projection of the Retina upon the Primary Optic Centers in the Rat, J. Comp. Neurol. **59**:341-373, 1934.

29. Bishop, G. H., and O'Leary, J. S.: Electrical Activity of the Lateral Geniculate Body of Cats Following Optic Nerve Stimuli, J. Neurophysiol. **3**:308-322, 1940.

30. Barris, R. W.; Ingram, W. R., and Ranson, S. W.: Optic Connections of the Diencephalon and Midbrain of the Cat, J. Comp. Neurol. **62**:117-154, 1935.

In man von Gudden [31] found that the finer fibers are connected with the superior colliculus and Winkler [6] that they penetrate the stratum opticum of the contralateral superior colliculus.

The Ventral Nucleus of the Lateral Geniculate Body.—The ventral nucleus of the lateral geniculate body is more primitive than the dorsal nucleus. In reptiles (Huber and Crosby [32]) it appears to be a differentiation of the thalamic tegmentum and receives terminals of the optic tract. In turn, fibers from the ventral nucleus end in the zona incerta and in the midbrain tectum. Woollard [33] identified this nucleus in Tupaia, Tarsius, Nycticebus and Hapale. Five years ago Balado and Franke [34] described it as composed of two parts, a loose and a thick division, not only in man but in two of the great apes, the chimpanzee and the orang-utan, and in several new world monkeys. The loose division resembled the zona incerta of the thalamus, and the thick division, they said, might be related to the optic tract. The ventral nucleus, the corpus praegeniculatum of human neurology, was described by Minkowski [35] as suffering transneuronal degeneration after long-standing enucleation of one eye. Balado and Franke, purely on anatomic grounds, stated the belief that nerve fibers pass out from this nucleus into the optic radiations. That there is some grave error in this interpretation is suggested by the recent studies of Marchi preparations made after removal of one eye in cats and in ferrets. In the first place, neither Barris, Ingram and Ranson [30] nor Barris [36] was able to trace any degenerating optic tract neurons into that nucleus in the cat; nor was Jefferson [22] able to find any in the ferret. In the second place, Barris and his co-workers found no retrograde degeneration within the ventral nucleus subsequent to removal of the area striata in the cat. On the other hand, as might have been expected, Bodian [21] found a few optic

31. von Gudden, B.: Ueber die Sehnerven, den Sehtractus, das Verhältnis ihrer gekreuzten und ungekreuzten Bündel, ihre Seh- und Papillarfasern und die Centren der letzteren, Tagebl. d. Versamml. deutsch. Naturf. u. Aerzte **58**:136, 1885.

32. Huber, G. C., and Crosby, E. C.: On Thalamic and Tectal Nuclei and Fiber Paths in the Brain of the American Alligator, J. Comp. Neurol. **40**:97-228, 1926.

33. Woollard, H. H.: Notes on the Retina and Lateral Geniculate Body in Tupaia, Nycticebus, and Hapale, Brain **49**:77-109, 1926.

34. Balado, M., and Franke, E.: Das Corpus Geniculatum externum: Eine anatomisch-klinische Studie, Berlin, Julius Springer, 1937.

35. Minkowski, M.: Ueber den Verlauf, die Endigung, und die zentrale Repräsentation von gekreuzten und ungekreuzten Sehnervenfasern bei einigen Säugetieren und beim Menschen, Schweiz. Arch. f. Neurol. u. Psychiat. **6**:201-252, 1920; **7**:268-303, 1920.

36. Barris, R. W.: Disposition of Fibers of Retinal Origin in the Lateral Geniculate Body: Course and Termination of Fibers of Optic System in Brain of the Cat, Arch. Ophth. **14**:61-70 (July) 1935.

tract terminals within the ventral nucleus in the opossum, and Packer [20] discovered a few of the crossed fibers entering this nucleus from the medial division of the optic tract in the marsupial phalanger Trichosurus vulpecula. But removal of the area striata in this animal was not followed by retrograde degeneration within the ventral nucleus as it was in the dorsal nucleus. Indeed, the ventral nucleus of the lateral geniculate body seems to be a primary optic center only in certain lower forms.

The Dorsal Nucleus of the Lateral Geniculate Body.—The dorsal nucleus of the lateral geniculate body has received more detailed study than any other primary optic center. Before discussing the anatomy of this nucleus, it is well to consider the retinal origin of the nerve fibers which travel in the tractus opticus. Polyak [37] described six morphologically distinct types of ganglion cells in the retina of man and of the macaque. The monosynaptic ganglion cell was related to a single bipolar cell, which in turn formed no synapse with a single cone cell. This ganglion cell had a thin slender axon and predominated in the fovea centralis and within the retinal areas immediately surrounding the fovea. The remaining five types of ganglion cells were polysynaptic, or diffuse. Three varieties of the diffuse types of ganglion cells (M, N, R) had thick axis-cylinders, and two varieties (O, P) sent off either medium-sized or thin axis-cylinders. In general the less diffuse types of the polysynaptic ganglion cells were those which had the thinner axis-cylinders and were more numerous in the more centrally located areas of the retina than in the peripheral zones. Further, the number of ganglion cells was greater in the central zones of the retina than in the peripheral regions. From these data it is safe to conclude (1) that the number of nerve fibers within the optic nerve per unit area of retina is greater for the fovea centralis and the neighboring central areas than for the peripheral parts of the retina and (2) that the majority of the thin and medium-sized fibers originate from the fovea itself or the zones surrounding the fovea. Consequently, both in the monkey and in man small fibers must enter the lateral geniculate body.

The first attempt to localize the terminals of retinal axons within the lateral geniculate body of mammals was made by Brouwer and Zeeman. Zeeman made small localized lesions in the retina of rabbits, cats and monkeys, and Brouwer and co-workers [27] determined the sites of degeneration with the Marchi technic. They reported that the retinal nerve fibers from the lower quadrants were projected on the dorsal half and those from the upper quadrants on the ventral half of the opposite lateral geniculate body in both the rabbits and the cats; that the temporal half was medial and the dorsal half lateral, and that the uncrossed fibers projected similarly on the ipsilateral nucleus. In the

37. Polyak, S. L.: The Retina, Chicago, University of Chicago Press, 1941.

monkeys the localization of optic tract terminals resembled that previously described in part by Rönne [16] and Henschen [17] for man. The greater volume of the lateral geniculate body entertained the macular bundle, axis-cylinders from the upper quadrants of the macula being projected medially and from the lower quadrants laterally throughout all except the most caudal and rostral levels. The peripheral retinal nerve fibers entered the ventral part of the dorsal nucleus, the extreme peripheral part lying most ventrally, the dorsal divisions of the peripheral fibers ending medially and the ventral divisions ending laterally.

Although the dorsal nucleus of the lateral geniculate body is present in all mammals, it is laminated only in those forms which possess binocular vision and which have large numbers of uncrossed nerve fibers in the optic tract. The simple three-layered laminations of carnivores give way to a variety of complex laminar forms in primates. The significance of these variations in laminar patterns in primates is obscure (Le Gros Clark [38]). They are less highly developed in new world monkeys than in old world monkeys.

The large-celled elements are found in all primates and the small-celled elements in some primates. In the macaque (Le Gros Clark [39]) layers 1 and 2 are large celled and the remaining 4 small celled. The ratio of large cells to small cells in the monkey is 1 to 7 or 1 to 8; in man this ratio is slightly higher, 1 to 5 or 1 to 6. Henschen [40] distinguished the large cells as those which received the axons of peripheral vision and the small as those which entertained the terminals of macular vision. All six layers (Le Gros Clark [39]) are found only in the macular regions, and layers 1 and 2 are located in the area allocated by Brouwer and Zeeman to peripheral vision. Moreover, a few cells within the large-celled lamina must be related to macular vision, because a small circumscribed lesion of the macular region of the area striata (Le Gros Clark [39]) was followed by a sharply defined retrograde degeneration in the small-celled lamina in the rostral part of the lateral geniculate body and in laminas 1 to 5 in the caudal division of that nucleus. The atrophied area in the small-celled lamina was sharply defined, but the boundaries of the atrophied area within the large-celled lamina overlapped the normal areas. Similar experimental lesions made by Poljak [41]

38. Clark, W. E. L.: Lateral Geniculate Body in Platyrrhine Monkeys, J. Anat. **76**:131-140, 1941.

39. Clark, W. E. L.: The Laminar Organization and Cell Content of the Lateral Geniculate Body in the Monkey, J. Anat. **75**:419-433, 1941.

40. Henschen, S. E.: Ueber spezifische Lichtsinn- und Farbensinnzellen im Gehirn, Acta psychiat. et neurol. **6**:347-358, 1931.

41. Poljak, S.: Projection of the Retina upon the Cerebral Cortex, Based upon Experiments with Monkeys, A. Research Nerv. & Ment. Dis., Proc. **13**:535-557, 1934.

in the upper lip of the calcarine fissure produced retrograde degeneration in layers 1 to 5 in the medial division of the lateral geniculate body (the upper retinal quadrants), but one made in the lower lip of that fissure, although located in the lateral division of this nucleus, also involved the sixth layer.

Only one morphologically distinct type of cell was found in either the large-celled or the small-celled lamina. No intercalated short axon type of cell, so characteristic of the cat's lateral geniculate body (O'Leary [42]), existed in the monkeys studied by Le Gros Clark and by Polyak or in the chimpanzees reported on by Polyak and Hayashi,[43] for complete removal of one area striata in either of these primates was followed by complete degeneration of the ipsilateral geniculate body. No nerve cells are present in that nucleus which relate it to any subcortical center or which project to any cortical surface other than the area striata. Indeed, in spite of the intercalated cells in the lateral geniculate body of the cat (O'Leary [42]), all efferent fibers are corticipetal. No axon of a cell body lying in this nucleus in the cat terminates in the superior colliculus, the pretectal nucleus, the pulvinar or any other thalamic nucleus (Barris [36]). These data make it difficult to credit the presence of a genitotectal tract as described for man by Balado and Franke.

A similar exquisite anatomic localization of the axons of retinal cells extends to the lamina. Le Gros Clark and Penman [44] found that small lesions made within the macula were projected to discrete areas of one lateral geniculate body in a slender band of degeneration involving layers 2, 3 and 5 of the ipsilateral nucleus, and layers 1, 4 and 6 of the heterolateral one, in such close corresponding areas that Le Gros Clark [39] concluded that homonymous areas are represented within these bands. That the nerve cells in a slender band through the laminations are an anatomic unit was demonstrated by the result of injury to 1 sq. mm. of area 17 of the occipital lobe (Poljak [45]). The retrograde degeneration affected in radial fashion all six laminas. Small areas, then, on the cortical surface of the area striata must receive nervous impulses which arise from stimulation of corresponding points on the retina of each eye.

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42. O'Leary, J. L.: A Structural Analysis of the Lateral Geniculate Nucleus of the Cat, J. Comp. Neurol. **73**:405-430, 1940.

43. Polyak, S., and Hayashi, R.: The Cerebral Representation of the Retina in the Chimpanzee, Brain **59**:51-60, 1936.

44. Clark, W. E. L., and Penman, G. G.: The Projection of the Retina in the Lateral Geniculate Body, Proc. Roy. Soc., London, s.B **114**:291-313, 1934.

45. Poljak, S.: A Contribution to the Cerebral Representation of the Retina, J. Comp. Neurol. **57**:541-617, 1933.

But the most astonishing finding concerning neuronic relationships within the primary thalamic center was that of Glees and Le Gros Clark.[46] After the optic nerve in monkeys was sectioned, each degenerating optic nerve fiber was observed to terminate in a spray of five or six branches. Each branch ended as a degenerating *bouton* on one cell body within the lateral geniculate nucleus, and no geniculate nerve cell entertained more than one *bouton*. This is the only instance so far reported in which a nerve cell was found to be dependent on a single synapse for its stimulation. The condition is, however, not true for the cat. For in that animal (Glees [47]) a single optic fiber supplies as many as ten cells with end feet, and each principle cell of the thalamic nucleus entertains as many as forty *boutons*. No one knows what synaptic relationships exist in this nucleus in man, although Balado and Franke stated the belief a 1 to 1 relationship between optic nerve fibers and cells in the lateral geniculate body exists.

These data allow one conclusion. The dorsal nucleus of the lateral geniculate body is the primary thalamic center for sight, the nucleus of origin of corticipetal fibers only, in the cat, the macaque and the chimpanzee. At the present time there are no data which contradict Balado and Franke's description of the presence of a geniculotectal tract in man.

The Pulvinar.—The evidence which gave the pulvinar its classification as a primary optic center was purely anatomic. When dissected with an orangewood stick or studied in Weigert preparations, the optic tract appears to penetrate the pulvinar. Nevertheless, enucleation of one eye or cutting of the optic tract was not followed either by degeneration of myelin within the nucleus or by shrinkage or disappearance of nerve cells within this body. No geniculopulvinar tract has been found in the cat or the monkey, and it is extremely unlikely that such a system exists in man. Rioch [48] described a connection between the posterior part of the pulvinar and the optic tectum in carnivores, while Foix and Nicolesco [49] on anatomic grounds traced medullated fibers via the posterior commissure between the pulvinar and the contra-

46. Glees, P., and Clark, W. E. L.: The Termination of Optic Fibers in the Lateral Geniculate Body of the Monkey, J. Anat. **75**:295-308, 1941.

47. Glees, P.: The Termination of the Optic Fibers in the Lateral Geniculate Body of the Cat, J. Anat. **75**:434-440, 1941.

48. Rioch, D. M.: Studies on the Diencephalon of Carnivora: III. Certain Myelinated Fiber Connections of the Diencephalon of the Dog (Canis Familaris), Cat (Felis Domestica), and Aevisa (Crossarchus Obscurus), J. Comp. Neurol. **53**:319-388, 1931.

49. Foix, C., and Nicolesco, J.: Anatomie cérébrale: Les noyaux gris centraux et la région mésencéphalo-sous-optique suivi d' un appendice sur la maladie de Parkinson, Paris, Masson & Cie, 1925.

lateral tectum, the oculomotor nucleus and the medial longitudinal fasciculus. By the method of retrograde degeneration the pulvinar of the monkey was found to send axons to areas 7 and 22 (Le Gros Clark and Northfield [50]) and to area 19 (Walker [51]); that of the chimpanzee, to these three areas and to area 5 (Walker [52]). Dejerine [53] described a homologous pulvinocortical system which terminated not only in these areas but in the supramarginal and angular gyri of man.

Sections of the pulvinar stained by the Weigert method seem to show neuronic connections with neighboring thalamic nuclei. These connections have not been tested by degeneration methods. The increase in absolute and in relative size of this nucleus in primates, particularly in man, as well as its projection on the lateral surface of the region where the higher association centers of the three posterior lobes converge, suggests a function of complex correlation of tactile sensibility with muscle sense, of visual images with movements of the eyes.

The two primary optic centers common to all mammals are the superior colliculus and the dorsal nucleus of the lateral geniculate body. The direct projection of optic tract fibers into the tegmentum of the midbrain and of the thalamus and into the ventral nucleus of the lateral geniculate body is a primitive condition.

THE CORTICAL CENTERS FOR SIGHT

The analysis of projection of the retina on the calcarine cortex in monkeys (Brouwer, Van Heuven and Biemond [54]; Van Heuven [55]) demonstrated that peripheral vision is located in the calcarine fissure and macular vision on the lateral surface of the occipital lobe, the dorsal quadrants above and the ventral quadrants below. By similar methods Walker and Fulton [56] concluded that the medial half of the lateral geniculate body projects to the upper lip of the calcarine fissure in the chimpanzee, the posterior part to the caudal pole and the anterior part

50. Clark, W. E., and Northfield, D. W. C.: The Cortical Projection of the Pulvinar in the Macaque Monkey, Brain **60**:126-142, 1937.

51. Walker, A. E.: The Primate Thalamus, Chicago, University of Chicago Press, 1938.

52. Walker, D. E.: The Thalamus of the Chimpanzee, J. Anat. **73**:37-93, 1938.

53. Dejerine, J.: Anatomie des centres nerveux, Paris, J. Rueff, 1901.

54. Brouwer, B.; van Heuven, G. J., and Biemond, A.: Experimentell-anatomische Untersuchungen über die optischen Systeme im Gehirn, Verhandel. v. k. Akad. v. Wetensch. **31**:603-619, 1928.

55. van Heuven, G. J.: Experimenteel-anatomisch onderzoek omtrent de corticale optische projectie bij den Java- aap, Inaug. Dissert., Amsterdam, 1929; abstracted, Am. J. Ophth. **13**:929, 1930.

56. Walker, A. E., and Fulton, J. F.: The Thalamus of the Chimpanzee, Brain **61**:250-268, 1938.

to the more oral division. The results of this incomplete analysis suggest a projection on the area striata of this animal which resembles that given by Holmes [14a] for man.

Within the area of macular vision on the lateral surface of the occipital lobe of monkeys, Talbot and Marshall [57] were able to locate a macular pattern by the electrical method of determining localization of response to physiologic stimuli. The half-fovea-centralis was projected to an area 6 mm. in radius situated 5 mm. below the tip of the sulcus lunatus. And spreading posteriorly in a family of curves, each degree of the macula was projected on strips of the area striata 3 mm. in width. The right horizontal meridian cut these strips in the vicinity of the line Van Heuven and Polyak had indicated as dividing the dorsal and the ventral macular quadrants. The visual fields being translated into retinal fields, the upper vertical nasotemporal line followed the outline of area 18, and the lower line disappeared within the inferior occipital sulcus. This large cortical surface is set aside for the reception of impulses originating from two half-retinal fields 3 mm. in diameter. No evidence for bilateral representation of this area was found.

Is man in this characteristic also not a monkey? Certainly, with the surgical removal of one occipital lobe in man bilateral representation of the macula returned. The old explanations for the sparing of central vision reappeared and was attacked by various investigators. Brouwer [58] reported a lesion limited to the root of the upper lip of the left calcarine fissure with normal central vision. The retrograde degeneration was localized in the area of peripheral vision within the ipsilateral geniculate body, and he concluded that macular vision is projected throughout the whole area striata. Theoretically, he should have found some retrograde atrophy in the macular area of the lateral geniculate body. Penfield, Evans and MacMillan [59] explained macular sparing subsequent to removal of one occipital lobe by the presence of collaterals from the macular bundle of the geniculo-occipital radiation which pass via the corpus callosum to the opposite occipital lobe. Yet no one has seen these collaterals (Putnam [60]), nor has any one proved that fibers from

57. Talbot, S. A., and Marshall, W. H.: Physiological Studies on Neural Mechanisms of Visual Localization and Discrimination, Am. J. Ophth. **24**:1255-1263, 1941.

58. Brouwer, B.: Projection of the Retina on the Cortex in Man, A. Research Nerv. & Ment. Dis., Proc. **13**:529-534, 1934.

59. Penfield, W.; Evans, J. P., and MacMillan, J. A.: Visual Pathways in Man, with Particular Reference to Macular Representation, Arch. Neurol. & Psychiat. **33**:816-834 (April) 1935.

60. Putnam, T. J.: Studies on the Central Visual Connections: General Relationship Between External Geniculate Body and Visual Cortex in Man, Arch. Neurol. & Psychiat. **16**:566-596 (Nov.) 1926.

one area striata pass to the opposite area striata through the corpus callosum in man (Van Valkenburg [61]) or in the monkey (Biemond [62]), as they do in the cat (Polyak [63]). Retrograde degeneration subsequent to a lesion of the occipital lobe has been found solely in the ipsilateral geniculate body. Foerster [64] removed the right occipital lobe and cut the corpus callosum, in searching for a tumor of the tectum, and left the patient with a complete left homonymous hemianopsia, due, he thought, to the latter procedure. As Horrax and Putnam [65] justly pointed out, the result might have been similar without cutting of the neopallial commissure. However, Hyndman [66] resected the right occipital lobe, and homonymous hemianopsia with sparing of central vision resulted; subsequent incision of the corpus callosum (5.7 cm.) was not credited with the subsequent encroachment on the macula. Hyndman's conclusion resembled Brouwer's.

At last a case had appeared in which ablation of the posterior part of the occipital lobe caused complete homonymous hemianopsia! To make matters more complex, of the 2 patients studied by Halstead, Walker and Bucy,[67] the one with the greater removal had sparing of central vision for color and form as well as for brightness, and the one with the smaller removal, a loss of these three elements of vision passing perpendicularly through both foveas. These men offered no explanation. They recalled Wilbrand's [68] reports of sparing of central vision associated with lesions of the optic tract with the explanation of bifurcation of fibers in the optic chiasma. Such a bifurcation has not been reported in monkeys. Polyak [37] stated the belief that the retinal ganglion cells

61. Van Valkenburg, C. T.: Experimental and Pathologico-Anatomical Researches on the Corpus Callosum, Brain **36**:119-192, 1913.

62. Biemond, A.: Experimentell-anatomische Untersuchungen über die corticofugalen optischen Verbindungen bei Kaninchen und Affen, Ztschr. f. d. ges. Neurol. u. Psychiat. **129**:65-127, 1930.

63. Polyak, S.: An Experimental Study of the Association Callosal and Projection Fibers of the Cerebral Cortex of the Cat, J. Comp. Neurol. **44**:197-258, 1927.

64. Foerster, O.: Beiträge zur Pathophysiologie der Sehbahn und der Sehsphäre, J. f. Psychol. u. Neurol. **39**:463-485, 1929.

65. Horrax, G., and Putnam, T. J.: Distortions of the Visual Fields in Cases of Brain Tumour: Field Defects and Hallucinations Produced by Tumours of Occipital Lobe, Brain **55**:499-523, 1932.

66. Hyndman, O. R.: The Central Visual System: Evidence Against Bilateral Representation Through Splenium of Corpus Callosum, Arch. Neurol. & Psychiat. **42**:735-742 (Oct.) 1939.

67. Halstead, W. C.; Walker, A. E., and Bucy, P. A.: Sparing and Non-Sparing of "Macular" Vision Associated with Occipital Lobectomy in Man, Arch. Ophth. **24**:948-966 (Nov.) 1940.

68. Wilbrand, H.: Ueber die makuläre Aussparung, Ztschr. f. Augenh. **58**:261-268, 1926.

give off no collaterals in man. It is true that von Kölliker [69] and Ramón y Cajal [70] have seen such a bifurcation in the chiasma of rabbits and of cats. Certainly, cutting only the corpus callosum in human beings produces no change in the visual fields. Akelaitis [71] studied with meticulous care 6 patients of Van Wagenen both before and after the corpus callosum was completely severed. No change whatever in any element of vision, from its simplest to its most complex, including apraxia and aphasia, followed this procedure.

When the evidence for the allocation of retinal projection within the optic radiations is considered, an explanation of macular sparing is still wanting. Polyak [37] promised a companion volume to "The Retina," in which, he said, he will prove that central vision lies in the horizontal meridian of the superior longitudinal fasciculus, the external sagittal stratum, the upper quadrants above and the lower quadrants below, i. e. a horizontal layering proceeding dorsally and ventrally from the perifoveal zones. Both Winkler [72] and Putnam [60] located the upper and lower quadrants within the upper and lower divisions of the external sagittal stratum. Neither gave central vision a discrete localization within this projection system. If the macular fibers are always arranged in the optic radiations in the order suggested by Polyak, it is logical to ask why some degree of semicircular bilateral scotoma did not result from destruction of the middle part of the stratum sagittale externum in the case reported by Holmes.[15a] Is it possible that individual variation in localization of the fibers representing central vision exists in the geniculo-occipital projection?

Certainly removal of the posterior part of the occipital lobe should give bisection of the macula if it was projected solely on the posterior third of the area striata. The recent findings with one exception are against such a localized projection. Macular vision escapes. And it is good that this is so, even if there is no adequate explanation.

CORTICIFUGAL AND ASSOCIATIONAL CONNECTIONS OF THE OCCIPITAL LOBE

The occipital lobe of man is related to the frontal, temporal and parietal lobes, to the gyrus angularis and to the precuneus in particular

69. von Kölliker, A.: Chiasma Opticum, 1899; cited by Minkowski.[35]

70. Ramón y Cajal, S.: Histologie du système nerveux de l'homme et des vertebrés, Paris, A. Maloine, 1911.

71. Akelaitis, A. J.: Studies on the Corpus Callosum: II. The Higher Visual Functions in Each Homonymous Field Following Complete Section of the Corpus Callosum, Arch. Neurol. & Psychiat. **45**:788-796 (May) 1941.

72. Winkler, C.: On Localized Atrophy in the Lateral Geniculate Body Causing Quadrantic Hemianopsis of Both the Right Lower Fields of Vision, Folia neurobiol. **7**:1-22, 1913.

by sheets of fibers, which have been dissected. The occipitopontile system arises from the lateral surface of the frontal lobe, presumably area 19, and the occipitotectal from the medial surface of this area (19) as well. The occipitotectal system penetrates both the outer superficial fiber stratum and the inner stratum opticum. Besides these incoming fibers, Winkler [6] has described a tecto-occipital system which originates within the third cellular layer. No corticifugal system from area 17 or area 18 has been described for man. Yet the infragranular layers of these two cortical areas are well developed. These are the layers from which corticifugal systems are thought to arise, for the supragranular layers, with perhaps the exception of the cells in layer IIIc, have been interpreted as the origin of both intraregional and interregional axons (Hines [73]).

Judging by the degeneration of myelin, the medial division of the area striata in the monkey is related to all neighboring regions, to the whole of the parietal lobe and to the precentral gyrus; the lateral surface of the area striata, to the whole area striata. These findings of Mettler [74] were challenged by Le Gros Clark,[75] for if a lesion in the cortex of a monkey involved the cellular layers only, the greatest length of an interregional association fiber on the lateral surface of the area striata did not exceed 5 mm. Although his lesions were large, Biemond [62] was unable to establish extension of the association fibers beyond the parietal lobe.

Mettler has found corticotectal systems from areas 18 and 19 and probably from area 17 as well; a corticopontile system (Biemond [62]) was found subsequent to a lesion which involved all three areas. Brouwer [76] and Biemond [62] stated the belief that they had demonstrated that each quadrant division of the lateral geniculate body of the monkey receives corticifugal fibers from that part of area 17 in which geniculo-occipital axons terminate. Le Gros Clark mentioned no such system, and Barris [36] said definitely that no corticogeniculate tract exists in the cat, but rather an occipitotectal and an occipitopontile tract and one to the nucleus of the optic tract. Is there no corticogeniculate tract in man, as there is in the monkey? Or have the conditions not been right for its discovery?

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73. Hines, M.: Cyto-Architecture of the Cerebral Cortex in Man, A. Research Nerv. & Ment. Dis., Proc. **13**:26-38, 1934.

74. Mettler, F. A.: Corticifugal Fiber Connections of the Cortex of Macaca Mulatta: Occipital Region, J. Comp. Neurol. **61**:221-256, 1935.

75. Clark, W. E. L.: Observations on the Association Fibre System of the Visual Cortex and the Central Representation of the Retina, J. Anat. **74**:225-235, 1941.

76. Brouwer, B.: Ueber die zentrifugale Beeinflussung von zentripetalen Systemen in Zentralnervensystem, Deutsche Ztschr. f. Nervenh. **105**:9-21, 1928.

RESULTS OF ELECTRICAL STIMULATION

As was to be anticipated from the fiber connections, Walker and Weaver [77] elicited in the monkey some variety of conjugate movements of the eyes from each division of the occipital lobe. On the other hand (Krause [78] and Foerster [79]), electrical stimulation of area 17 in a conscious human being has not produced any motor effect in the hands of any investigator. Rather, stimulation of the posterior part, where macular vision is located, elicited a stationary light or stationary stars in front of the eyes; of the anterior part (peripheral vision), light in the peripheral field moving toward the center; of the superior lip, light from below; of the inferior lip, light from above. Stimulation of area 18 did not elicit movements of the eyes as it might have been expected to do from the cytoarchitecture; instead, it produced optic sensations, such as flames, stars and rings, while stimulation of area 19 caused conjugate deviation of the eyes to the opposite side and optic phenomena. These phenomena were hallucinations, such as lights, colors, figures, little people and animals. Penfield and Evans,[80] unlike Foerster, have never produced definite forms, only colors, by stimulation of area 19.

RESULTS OF INJURY

Injury to the Primary Visual Centers.—In man lesions of the superior colliculus are accompanied not by any loss of vision but rather by loss of the pupillary response to light; in the cat and in the monkey the pretectal area must be included. Partial lesions of one lateral geniculate body cause bilateral symmetric scotomas; complete lesions, homonymous hemianopsia passing vertically through central vision. Henschen [81] has said that lesions of the pulvinar were not accompanied by loss of vision. Winkler [82] described a tumor which annihilated the pulvinar and did not injure the lateral geniculate body but unfortunately severed some of the optic radiations. Instead of bilateral symmetric scotomas for all aspects of vision, there resulted homonymous hemianopsia for form only!

Injury to the Cortical Projection Center.—Injury to the whole area striata in man produces complete loss of visual sensations, but not loss

77. Walker, A. E., and Weaver, T. A.: Ocular Movements from the Occipital Lobe in the Monkey, J. Neurophysiol. **3**:353-357, 1940.

78. Krause, F.: Die Sehbahn in chiurgischer Beziehung und die Faradische Reizung des Sehzentrums, Klin. Wchnschr. **3**:1260-1265, 1924.

79. Foerster, O.: The Cerebral Cortex in Man, Lancet **2**:309-312, 1931.

80. Penfield, W., and Evans, J.: Functional Defects Produced by Cerebral Lobectomies, A. Research Nerv. & Ment. Dis., Proc. **13**:352-377, 1934.

81. Henschen, S. E.: Sur le centre cortical de la vision, Cong. internat. de méd. Rap. sect. d'opht., 1900, pp. 232-249.

82. Winkler, C.: A Tumor in the Pulvinar Thalami Optici: A Contribution to the Knowledge of the Vision of Forms, Verhandl. v. k. Akad. v. Wetensch. **13**: 928-938, 1911.

of visual memory or of visual orientation (Ingham [83]), with retention of reflex constriction of the pupil to light and to movements of convergence and with retention of normal motility of the eyeball. Similarly, the dog, the cat and the monkey become blind. Each of them is unable to avoid objects. Nevertheless, Marquis [84] has demonstrated that the ability to form a stable habit of light discrimination remained in dogs after ablation of the cortical area for sight. The threshold, however, was raised. With Hilgard, Marquis [85] reported that 1 of 3 monkeys retained a visually conditioned response after removal of both occipital lobes. Klüver [86] found that 2 monkeys from which both occipital lobes had been removed learned to distinguish the box containing food after twenty minutes of dark adaptation by differences in relative intensity of light and in position of lights; but they found little evidence for the effectiveness of configuration as a stimulus, none at all for the effectiveness of color and no response in daylight conditions. All successful tests had to be carried out either in the dark or under conditions of low illumination. In consideration of the similarity in structure of the retina and of the relation of the retina to the lateral geniculate body and to the area striata, it is peculiar that the physiologic state of the monkey is so dissimilar to that of man. On the other hand, has any one ever required a cortically blind human being to secure his food by distinction between lights in a dark room? Or is the ability of the monkey's midbrain tectum to make distinction between relative intensities of light being recorded? Or were those 2 monkeys utilizing another part of the cortex, reached via a route at present unknown, a part of the cortex which might be in some manner homologous to man's supramarginal gyrus? For Penfield and Gage [87] stated the belief "that neuronal discharge in the region of the supramarginal gyrus and possibly extending into areas 19 and 22 may produce a sensation of light in the absence of the area striata."

Injury to the Higher Cortical Association Centers for Sight.—Lesions described some twenty-five years ago (Hines [88]) producing disturbances of spatial orientation and localization of objects in the visual fields were frequently bilateral injuries to the supramarginal or angular gyri, which

83. Ingham, S. D.: The Functional Relations of the Primary, Secondary and Tertiary Visual Cortical Areas, Bull. Los Angeles Neurol. Soc. **5**:12-17, 1940.

84. Marquis, D. G.: Effects of Removal of the Visual Cortex in Mammals, with Observations on the Retention of Light Discrimination in Dogs, A. Research Nerv. & Ment. Dis., Proc. **13**:558-592, 1934.

85. Marquis, D. G., and Hilgard, E. R.: Conditioned Responses to Light in Monkeys After Removal of Occipital Lobes, Brain **60**:1-12, 1937.

86. Klüver, H.: Visual Functions After Removal of the Occipital Lobes, J. Psychol. **11**:23-45, 1941.

87. Penfield, W., and Gage, L.: Cerebral Localization of Epileptic Manifestations, A. Research Nerv. & Ment. Dis., Proc. **13**:593-613, 1934.

88. Hines, M.: On Cerebral Localization, Physiol. Rev. **9**:508-517, 1929.

according to the nature of the wound must have injured cortical radiations of the pulvinar. In cases in which only one inferior parietal lobule was injured, the wound was deep enough to destroy the optic radiations and therefore may have severed some of the pulvinocortical projections. All the patients had in common not only difficulty of measuring and of localizing objects in space by sight but some type of disturbance of movements of the eyes, such as disturbance of fixation, convergence, accommodation and the following of a moving object. In some instances there was loss of the third dimension. As the patients themselves said, "Something else, that indicates depth, has been taken away" (Riddoch [89]) or, when offering an explanation for inability to see more than one side of an object, "My eyes couldn't get around the corner" (Holmes and Horrax [90]).

That such difficulties with movements of the eyes may be related to pulvinocortical projections rather than to cortical injury in the parietal lobe is further suggested by a recent report of Riddoch.[91] Riddoch studied 2 patients each of whom had a large tumor involving the lateral surface of the left parietal lobe, affecting only the more dorsal and posterior part of the inferior parietal lobule and almost the whole of the posterior part of the superior parietal lobule. Neither patient was aware of a spatial disorientation in the right homonymous half-fields, because both localization and estimation of distance were present in their left half-fields. After operation gross spatial disorientation, especially that of estimating absolute distance in the right half-fields, remained. Stereoscopic vision was intact. Visual fixation, pupillary accommodation, convergence and conjugate movements of the eyes returned. Both were able to reach accurately for near objects and to avoid obstacles. Apparently, visual disorientation can be present in a part of a visual field without disturbance in the remainder, without limitation of the visual fields themselves and without serious disturbance of central vision and can be dissociated from related disorders of visual function frequently found when the posterior part of the inferior parietal lobule is injured.

Brain [92] has recently published cases of two types of visual disorientation. The first type was similar to that of Riddoch in lesion and in symptoms; the second type was the result of lesions which transgressed

89. Riddoch, G.: Dissociation of Visual Perceptions Due to Occipital Injuries, Brain **40**:15-57, 1917.

90. Holmes, G., and Horrax, G.: Disturbances of Spatial Orientation and Visual Attention with Loss of Stereoscopic Vision, Arch. Neurol. & Psychiat. **1**:385-407 (April) 1919.

91. Riddoch, G.: Visual Disorientation in Homonymous Half-Fields, Brain **58**:376-382, 1935.

92. Brain, W. R.: Visual Disorientation with Special Reference to Lesions of the Right Cerebral Hemisphere, Brain **64**:244-272, 1941.

the occipital lobe also (right) and caused a left hemianopsia and neglect of the left half of external space. There was no loss of topographic memory and no inability to describe familiar routes in words; nevertheless the patient always lost his way by taking right instead of left turns even in his own house. In talking the left side of external space was there, and in walking it was not there.

No new contribution to the allocation of color vision to the lateral surface of the occipital lobe has appeared. Gelb and Goldstein's [93] association of loss of color vision with lesions of that area remains an isolated and unconfirmed finding. It is difficult to reconcile their reports with Henschen's [40] correlation of the presence of small cells in the lateral geniculate body and the area striata of monkeys with color vision or the postulation of Le Gros Clark [94] that the juxtaposition of three cellular elements in the retina, each of which terminates in three of the six laminas of the macular area of the lateral geniculate body, is the anatomic substrate for color vision. Nevertheless, color perception may be cortical. In 1933 Wechsler [95] described a patient in whom multiple lesions were undoubtedly present. Although he was able to perceive the form of objects when they were large, nevertheless he collided with them and groped for them. His color perception, however, was quick and accurate. He could pick out colors at command and distinguish even those he could not name. Yet he was a boy whose speech was unintelligible and whose apraxia could not be evaluated because of his astereognosis!

It is customary to associate injury to the dorsolateral part of one area 19 (the left in right-handed persons), particularly that portion which is contiguous with the parietal lobe, with losses in the highest visual sphere, the acquired symbolization of objects in visual fields. In 1937 Nielsen [96] summarized all unequivocal cases of visual agnosia for objects caused by a unilateral lesion of the occipital lobe. In 4 of the 13 cases the lesion was in the right occipital lobe. Three years later he [97] found 2 more cases, in which the lesion was the result of a vascular accident involving

93. Gelb, A., and Goldstein, K.: Ueber einige Ergebnisse farbenpsychologischer Analysen an Hirnverletzung, Deutsche Ztschr. f. Nervenh. **59**:216-225, 1918.

94. Clark, W. E. L.: Anatomical Basis of Color Vision, Nature, London **146**: 558-559, 1940.

95. Wechsler, I. S.: Partial Cortical Blindness with Preservation of Color Vision: Report of Case Following Asphyxia (Carbon Monoxide Poisoning?); Consideration of Question of Color Vision and Its Cortical Localization, Arch. Ophth. **9**:957-965 (June) 1933.

96. Nielsen, J. M.: Unilateral Cerebral Dominance as Related to Mind-Blindness: Minimal Lesion Capable of Causing Visual Agnosia for Objects, Arch. Neurol. & Psychiat. **38**:108-135 (July) 1937.

97. Nielsen, J. M.: Dominance of the Right Occipital Lobe, Bull. Los Angeles Neurol. Soc. **5**:135-145, 1940.

areas 18 and 19 of the right occipital lobe and the patient was right handed. One patient had visual agnosia for all objects and the other for inanimate objects only; both suffered from severe visual disorientation even in familiar surroundings. Brain [98] described the case (without autopsy) of a boy whose condition was a marked contrast both to that of Riddoch's and to that of Wechsler's patients and was similar in some respects to that of Nielsen's. The boy's estimation of absolute and of relative distances was good, but he became lost if he ventured more than a few hundred yards away from home. Although he had a fair but imperfect recognition for color, he had little or none either for objects or for pictures. He recognized his mother only when she spoke.

In a right-handed patient who recovered from apoplexy with a left-sided paralysis and mind blindness, Olsen [99] allocated to the destruction of the posterolateral part of areas 18 and 19 (right) the presence of visual agnosia for objects, persons (including her husband) and symbols; the loss of knowledge of the body self, and visual disorientation if familiar objects were moved. Retention of the capacity to recognize and name colors was interpreted as evidence of sparing of a small part of area 18 (right) or of the function of the minor (left) hemisphere. As a contrast to these findings of Olsen, German and Fox [100] found no mind blindness, rather a primary loss of verbalization in response to objects in the visual field, to follow ablation of the dominant (left) occipital lobe together with the posterior part of the parietal lobe.

Certainly it is clear that different moieties of vision can be injured differentially. It is uncertain whether color vision can be lost independently of loss for black and white, but color vision may be retained when that for form is severely disturbed. Loss of spatial orientation is not necessarily related to disturbance of movements of the eyes, while an ability to estimate distance may be present with severe disturbances of recognition of the significance of objects in the visual field.

Examination of the cortical surface allocated in mammals to the three divisions of the occipital lobe demonstrates that in all except man the area striata occupies the largest surface. And only in man has area 19 taken proportionately and absolutely the greatest surface. Reading Polyak's [37] description of the retina of the monkey in comparison with that of man did not convince me that the retina of man is superior. Indeed, if visual acuity is assessed by the degree of intermixture of rods and cones, by the density of conducting segments and by the extent and perfection of the

98. Brain, W. R.: Visual Object Agnosia with Special Reference to the Gestalt Theory, Brain **64**:43-62, 1941.

99. Olsen, C. W.: Mind-Blindness: Clinical and Pneumencephalographic Study of a Case of Infarction of the Brain, Bull. Los Angeles Neurol. Soc. **7**:43-47, 1942.

100. German, W. J., and Fox, J. C.: Observations Following Unilateral Lobectomies, A. Research Nerv. & Ment. Dis., Proc. **13**:378-434, 1934.

fovea, then some lower primates rank above man (Woollard[101]). Le Gros Clark[39] stated the belief that the lamination of the lateral geniculate body is more complicated, and the laminar differentiation of the area striata of a higher degree, in the macaque than in man. Indeed, the degree of anatomic differentiation within the optic pathways from retina to area striata as it is known seems to be greater in the macaque than in man. Certainly if a human being attempts to see the objects which excite minute investigation by the macaque, he will be more frequently disappointed than rewarded. Man differs from the macaque not on the level of objects seen but rather on that which endows the object in the field of vision with significance. He differs at the level of the mind's eye. It is probably significant that the area striata in all primates except man contains motor centers which move the eyeballs but in man these centers appear to be located not in the area striata itself but rather in the region of higher association. For looking is a prerequisite to seeing, and the ability to fix the eyes and to converge the eyes determines what objects will be examined in the visual field.

CONCLUSION

In conclusion, homonymous retinal half-fields are projected in an orderly manner (1) on the ipsilateral lateral geniculate body and (2) within the area striata, the greater part of each region being reserved for central vision. No evidence for bilateral representation of macular vision was discovered in the macaque, and no adequate explanation for its representation in man has been given.

In the cat, the macaque and the chimpanzee, all axons leaving the lateral geniculate body are corticipetal. The synaptic relations between the optic tract and the cells of the lateral geniculate body appear to provide for overlap in the cat and for simple multiplication in the macaque. These relations are unknown in the chimpanzee and in man.

Simultaneously with the appearance of the lateral geniculate body's exclusive projection to area 17, the retinal mosaic in the superior colliculus becomes less marked, and optic tract axons by-pass the ventral nucleus of the lateral geniculate body and become difficult to trace into the thalamic tegmentum. The optic system has not retained the phylogenetically old in the presence of greater differentiation within thalamic and cortical centers.

The fronto-occipital association bundles previously described for man are now denied for the monkey. Corticotectal, corticopontile and corticogeniculate fibers are recognized in the monkey and only the first two in the cat and in man. In man area 19 appears to be the sole origin of

101. Woollard, H. H.: The Differentiation of the Retina in the Primates, Proc. Zoöl. Soc. London **1**:1-17, 1927.

corticifugal systems as well as the only area in his occipital lobe which has yielded movements to electrical stimulation. Thus no terminations are known for the axons of the motor-like nerve cells in areas 17 and 18. Whatever may ultimately prove to be the condition in man, all association between the lateral geniculate body and the pulvinar must be routed through the midbrain tectum or the cortex cerebri in the cat and in the monkey.

A lesion of the lateral geniculate body or of the area striata in man is followed by a loss of visual sensations without disturbance of visual memory or visual orientation. Lesions of the remainder of the occipital lobe produce some type of visual disorientation. Four varieties are described: (1) a loss of ability to judge absolute distance in homonymous half-fields contralateral to a lesion in the posterior division of the parietal lobe, (2) a loss of estimation of distance for objects within arm's length or stepping distance, lesion unknown, probably in the parieto-occipital transition area, (3) a loss of one half of external space opposite to a lesion on the lateral surface of area 19 and (4) a loss of topographic memory and of the meaning of objects in the visual fields, allocated to a unilateral lesion of the peristriate area.

No new evidence for the cortical localization of color vision has been published, although color vision was found to be spared in persons who had lost some moiety of visual orientation. The presence of small cells in the lateral geniculate body of monkeys possessing polychromatic vision suggests the necessity of a central anatomic differentiation—a differentiation which may be concomitant rather than causal.

Moreover, a definite realization of the problems of localization of vision exists. With the increase in surgical skill and in neurologic education, certain gaps in the knowledge of localization of vision may be filled by cooperative endeavor between neurosurgeon and ophthalmologist. Further, the development of electrophysiology has necessitated new anatomic studies. A solution of anatomic problems alone will never provide an adequate or satisfactory understanding of vision. If the results of study of the optic system during the next decade are as fruitful as those of the last ten years, some present questions may have answers.

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Correspondence

CROSS CYLINDER AND ASTIGMATIC CHART

To the Editor:—Apropos of Dr. Wilson's instructive article on the cross cylinder and the astigmatic chart (Wilson, A.: Combination of Cross Cylinder and Astigmatic Chart in Correction of Astigmatism, ARCH. OPHTH. **28:** 490 [Sept.] 1942), the following considerations may be found helpful. There are a few points in the technic given which are not very clear, but the idea that one may use at option either plus or minus cylinders to equalize the clock lines is not always workable. In fact, whether to use plus cylinders or minus cylinders with the clock dial will depend on two factors: (1) whether the astigmatism is hyperopic or myopic, naturally or rendered so by auxiliary spheres, and (2) how the examiner wants to equalize the lines, that is, to equalize by clearing up the blurred lines or to equalize by blurring the clear lines. A fundamental principle in line chart tests is that the cylinder lens, plus or minus, affects primarily the clearness of the lines parallel to its axis. That is, a cylinder with its axis at 90, plus or minus, will affect the clearness of the 90 degree lines. A cylinder with its axis at 60 will affect the clearness of the 60 degree lines, that is, the clock lines running from 11 to 5, and so on.

Dr. Wilson says he prefers "to add plus lenses to the dark lines." If by this is meant that he uses plus cylinders with their axes parallel to the clearest (best defined) lines, then equalization of the lines will take place in simple or compound myopic astigmatism by a process of blurring the "dark" lines to the same extent as the "light" lines. In simple or compound hyperopic astigmatism, plus cylinders with axes parallel to the "dark" lines can also be made to equalize the lines by blurring the originally "dark" lines to the same extent as the "light" lines. But this will require an overcorrection, i. e., enough plus cylinder to create as much myopia in the meridian parallel to the "light" lines as there is hyperopia in the meridian parallel to the "dark" lines. This procedure is very much roundabout and is hardly workable.

More likely the statement "to add plus lenses to the dark lines" means to use plus cylinders with their axes at right angles to the "dark" lines. By this method equalization is possible only in cases of simple or compound hyperopic astigmatism, and equalization will take place by a process of clearing up the "light" lines. If the astigmatism is simple or compound myopic, plus cylinders with axes at right angles to the "dark" lines will never equalize the lines, either by clearing up the "light" lines or by blurring the "dark" lines.

A similar analysis can be applied to the use of minus cylinders. In general, one cannot simply choose to use at will either plus or minus cylinders for equalizing the lines. One must consider the nature of the refractive error and whether one wants to equalize by clearing the blurred lines or by blurring the clear lines. In hyperopic astigmatism, plus cylinders with axes parallel to the blurred lines (best located as at right angles to the clearest lines) will equalize by clearing the blurred

lines. Minus cylinders with axes parallel to the clear lines will equalize by blurring the clear lines. In myopic astigmatism, minus cylinders with axes parallel to the blurred lines will equalize by clearing the blurred lines. Plus cylinders with axes parallel to the clear lines will equalize by blurring the clear lines. In general, it is better to equalize by clearing up the blurred lines than by blurring the clear lines.

The cross cylinder can be used with the clock lines to check the accuracy of the amount of cylinder. The eye is slightly fogged, and a weak cross cylinder, say $+ 0.12 \bigcirc - 0.25$ D. cyl., is used and flipped in the two positions, say (1) with the minus cylinder at axis 90 and (2) with the minus cylinder at axis 180. If the cylinder is right in amount, then when it is in the first position the vertical lines will stand out "darker," and when it is in the second position the horizontal lines will stand out "darker" to the same extent. If the cylinder is not right in amount, then in one position the "darkening" either may not be noted at all or may appear less pronounced.

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Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Cornea and Sclera

THE AETIOLOGY OF PHLYCTENULAR OPHTHALMIA. A. SORSBY, Brit. J. Ophth. **26:** 159 (April) 1942.

This is the Hunterian Lecture of 1942.

The article begins with a review of the literature of phlyctenular ophthalmia. Statistical studies show that there has been a consistent decline in the number of cases of this disease in the United States and in several European countries. There are two exceptions: The data of the Hôtel Dieu, at Paris, disclose a practically continuous increase in the number of cases in the period 1912 to 1932, and the more important exception there was a consistently higher incidence reported in several centers during the first World War and its aftermath.

Phlyctenulosis is essentially a disease affecting children and young adults. Females are much more frequently affected than males. An increase in incidence in the spring has been noted by a number of observers.

Phlyctenulosis in tropical countries appears to have clinical and possibly, also, histologic features that differ from those seen in Europe and in the United States.

Among the attributed causes of the disease are nutritional deficiencies, focal infections, pediculosis capitis, endocrine disturbances and tuberculosis. Sorsby states: "Almost every possible fault in diet has been incriminated as the cause of phlyctenular ophthalmia. . . . Enlarged and septic tonsils are regarded by some observers as causal factors. . . . A growing volume of elaborate evidence is available on the significance of tuberculosis as the cause of phlyctenular ophthalmia. In so far as the tubercle bacillus has not been found in phlyctenulae, and in that the implantation of the organism in the conjunctiva produces a tuberculous reaction and not a phlycten, the evidence is negative, but a mass of observations is available implicating tuberculous infection rather than tuberculous disease. . . . Since the introduction of the von Pirquet test in 1907 and its subsequent modification, a high percentage of positive tuberculin reactions has been consistently reported by a number of observers."

S. H. MCKEE and W. ZENTMAYER.

THE AETIOLOGY OF PHLYCTENULAR OPHTHALMIA. A. SORSBY, Brit. J. Ophth. **26:** 189 (May) 1942.

In his continuation of the article which appeared in the April issue, Sorsby analyzes the cases of phlyctenular ophthalmia observed at the White Oak Hospital, London. During the sixteen years from 1925 to 1940, 1,392 children with this disease were admitted to the hospital. The records of those treated between June 1935 and December 1940

were investigated. The following summary by the author covers the interesting points disclosed:

"It is concluded that phlyctenular ophthalmia is a manifestation, not of tuberculous disease, but of tuberculous infection, the phlycten making its appearance only when a hyperallergic phase is present and there is a suitable (? specific) exciting factor of endogenous or exogenous origin. It is suggested that the age distribution, sex-incidence and seasonal variation seen in phlyctenulosis are best explained on the assumption that the affection is tuberculous in character. The prognostic significance of the phlycten is graver than that of a positive tuberculin reaction in a child; this is borne out by the subsequent incidence of tuberculosis and mortality from it in children previously affected with phlyctenulosis."

W. ZENTMAYER.

Experimental Pathology

LENS OPACITIES ASSOCIATED WITH EXPERIMENTAL CALCIUM DEFICIENCY. K. C. SWAN and P. W. SALIT, Am. J. Ophth. **24:** 611 (June) 1941.

Swan and Salit give the following summary of their experiments:

"Lens opacities developed in young rabbits placed on a calcium-deficient diet at the time of weaning. These opacities developed in association with low serum calcium and tetany, and could be prevented by the addition of calcium salts to the diet. Only the newly formed lens fibers appeared to be involved. Further studies will be required to determine the mechanism of formation of the opacities and modifying factors."

W. S. REESE.

General

THE STERILIZATION OF SHARP INSTRUMENTS BY CHEMICAL SOLUTIONS. M. H. POST, Am. J. Ophth. **24:** 624 (June) 1941.

Post remarks that proper sterilization and preservation are the two requisites in sterilization of sharp instruments. He considers heating and boiling less desirable than chemical sterilization. As a result of his studies he suggests the use of a solution of 2 parts each of cresol, formaldehyde and thymol and 94 parts of 95 per cent alcohol.

W. S. REESE.

General Diseases

KERATITIS DUE TO MUMPS. R. W. DANIELSON and J. C. LONG, Am. J. Ophth. **24:** 655 (June) 1941.

Danielson and Long draw the following conclusions:

"Descriptions of seven cases of keratitis following mumps have been found in the literature, and one new case has been observed by the writers. There is sufficient similarity in the onset, course, and appearance of these cases to justify considering this condition as a definite clinical entity. The disease affects one eye only and begins from 2 to 11 days after the onset of the parotid swelling. The cornea is rather uniformly grayish-white with very minute white interlacing fibrillae. Pain is not

severe, but photophobia and lacrimation are present. Vision is markedly reduced. Improvement occurs with astonishing rapidity, resulting in complete recovery within a month or less after onset. There is no vascularization nor scarring of the cornea. The uvea is apparently not involved in the inflammatory process."

W. S. REESE.

OCULAR SYMPTOMS IN PARADENOLYMPHITIS (VENEREAL LYMPHOGRANULOMA). A. VÁZQUEZ BARRIÈRE and J. MAY, Arch. de oftal. de Buenos Aires **16:** 1 (Jan.) 1941.

After an exhaustive study of the literature, Vázquez Barrière and May report on 120 cases of venereal lymphogranuloma in the secondary stage. The following ocular symptoms were found: biomicroscopic visibility and tortuosity of nerve filaments in the center of the cornea, blurring of the disk margins with a slight surrounding grayish retinal edema, dilatation and tortuosity of the retinal veins, ocular hypotension and retinal hypertension. The ocular lesions did not produce any permanent defect and were found in cases both of acute and of chronic infection.

Vázquez Barrière and May consider the ocular symptoms an aid to the diagnosis of this disease.

C. E. FINLAY.

Glaucoma

TREATMENT OF GLAUCOMATOUS DISEASES. E. P. FORTIN, Arch. de oftal. de Buenos Aires **16:** 316 (June) 1941.

The classic description of the iris, ciliary body and anterior chamber is faulty because it is based on the study of dead tissue in a state of advanced decomposition after lengthy maceration in a violently destructive fixation agent, heated to a degree which coagulates and destroys albuminous tissues, with artefacts substituted for the destroyed tissues. Fortin states that methods of examination (slit lamp examination, gonioscopy and endoscopy) exist which permit the examination of living tissues and eliminate the traps referred to.

In the region under study in glaucoma, artefacts give rise to unexplained physiology, a pectinate ligament which separates the anterior chamber from Schlemm's canal being described. In this area, Fortin claims to have discovered a remarkable filtering structure, which he calls an ocular kidney, which opens under the action of the ciliary muscle, special cells attached to the corneal lamellas presiding over the filtration. He insists that the iris does not contain connective tissue and that in it a cavity is found continuous with that of the ciliary processes, filled with a gelatinous tissue in which float fine vessels, nerve fibers and interesting chromatophore cells, no anatomic delimitation being found in a longitudinal section of the eyeball parallel to the optic nerve. The only fixation of the ciliary muscle to the corneoscleral envelope is a fine, resistant ring in the form of a canal, which contains Schlemm's canal, the only other attachment taking place through a few arterioles and the long posterior ciliary arteries, with no normal union existing between the scleral and the uveal envelope, an arrangement which allows free movement of the muscle. In chronic glaucoma, the dorsal surface of

the ciliary muscle should be carefully studied, pathologic adhesions and an excessive accumulation of fluid being found in gout and in rheumatism.

The author has also confirmed the existence of an irregular venous circle, or sinus, external to and 4 to 6 mm. behind the corneoscleral limbus, from which radiate numerous small veins, short anteriorly and long posteriorly. This venous sinus should be carefully studied in cases of glaucoma, as it constitutes the outlet of the fluid which escapes through Schlemm's canal, which is opened up by the ciliary muscle.

The current anatomic descriptions, moreover, omit the fact that the long posterior ciliary arteries pierce but do not run in contact with the ciliary muscle, blood flowing readily through them when the muscle is paralyzed or atropinized and its flow being retarded when the muscle is contracted by physostigmine, pilocarpine or strychnine. Veins are scarce in the ciliary body, being situated external to the muscle.

As can be seen from the foregoing discussion, there is today no satisfactory explanation for the generally prevalent views of the beneficial action of iridectomy and of miotics in glaucoma, it being assumed in regard to the latter that the minute iris sphincter on contracting opens the filtration angle, an angle which the author has elsewhere indicated does not exist, the only angle present at this site being that between the longitudinal and the circular ciliary muscle fibers, where no filtration outward is possible. The filtration from this anterior chamber to the conjunctiva occurs at the level of the pectinate filter represented by the ring posterior to Schlemm's canal, situated 1 mm. in front and internal to this angle (as illustrated by large photomicrographs). The author, further, refers to prior publications of his showing that the ciliary muscle has the following actions: closure of the nutritive arteries of the eye, especially the long posterior ciliary arteries; opening of the choroidal veins; opening of the pectinate filter and opening of Schlemm's canal through widening of the corneoscleral spaces, which act as an aspirating pump, drawing the ocular humors into the venous sinus situated external to the corneoscleral limbus. According to this theory it is important to instil miotics in several doses, as it is necessary in each individual case to determine the action of these drugs to be able to obtain their maximal effect.

In dealing with a case of glaucoma, before starting the treatment one should carefully determine the following points: the range of accommodation, the existence of astigmatism against the rule as indicative of an abnormal rotation of the lens on its vertical axis, the depth of the anterior chamber, the possible existence of pathologic adhesions between the ciliary body and the sclera, the condition of the extralimbal venous sinus, the existence of fluid between the sclera and the uvea, the ocular tension at different hours and the effect of fluorescein and of gonioscopy.

Gonioscopy can give fairly accurate information on the condition of the corneoscleral limbus. It should be employed before and after instillation of physostigmine salicylate, there being few cases in which the pectinate ring is totally obstructed. Exfoliation of the anterior lens capsule must also be looked for. The Kniess closure and inflammation of the pectinate ligament, although frequent, are not constant; in many cases both lesions are absent.

In connection with operations for glaucoma, Fortin stresses the extreme delicacy of the technic, a difference of 1 mm. in the location of the incision making a great difference. It is desirable that the patient be absolutely quiet and that the operator work under an amplification of 10 to 15 diameters. In iridectomy in the treatment of acute glaucoma, he considers the opening of the iridociliary sinus as the essential feature, it being followed by the escape of a yellowish liquid.

C. E. FINLAY.

Lacrimal Apparatus

STREPTOTHRICOSIS OF THE LACRIMAL CANALICULI. A. J. ELLIOT, Am. J. Ophth. **24:** 682 (June) 1941.

Elliot gives the following summary:

"Nine cases of streptothricosis of the lacrimal canaliculi are reported. All the patients were females, the majority of whom were between the ages of 46 and 58 years.

"Pus at the inner canthus and epiphora were the chief complaints. One patient had an acute unilateral conjunctivitis.

"In two of the cases, both upper and lower canaliculi were involved, while in two others, only the upper canaliculus was affected.

"The patients in whom concretions only were expressed were cured in two or three treatments, while when a mucopurulent material was obtained the treatment had to be prolonged.

"Treatment consisted of frequent dilation of the punctum, curettage, and manual expression of the canaliculus. Lugol's iodine [compound solution of iodine U. S. P.] was instilled into the canaliculus and potassium iodide was given by mouth."

W. S. REESE.

Lens

INVESTIGATION CONCERNING THE LENS CAPSULE: ITS IMPORTANCE IN THE TECHNIQUE OF INTRACAPSULAR CATARACT EXTRACTION. M. KLEIN, Brit. J. Ophth. **26:** 118 (March) 1942.

After discussing the anatomic and physiologic aspects of intracapsular extraction, Klein states that the conditions in the cataractous eye are generally favorable for intracapsular extraction.

W. ZENTMAYER.

A FIRST HUNDRED CASES OF INTRA-CAPSULAR CATARACT EXTRACTIONS. J. B. MCAREVEY, Brit. J. Ophth. **26:** 275 (June) 1942.

The lens was delivered in its capsule in 67 cases out of the 100. In 21 cases the capsule ruptured during the expression of the lens; in 4 cases the lens was intumescent and it was not possible to grasp it with the forceps. In 8 cases it was not possible to dislocate the lens with reasonable pressure and traction; so the intracapsular method was not persisted in. The procedure employed was that of Elschnig. The chief causes of rupture of the capsule were too small a section, faulty pressure with the hook and unnecessary traction with the forceps. There was loss of vitreous in 3 cases and expulsive hemorrhage in 1. One eye

was lost from panophthalmitis. The resulting visual acuity was 6/24 or better in 28 cases, 6/60 in 1, 4/60 in 1, light perception in 1 and blindness in 2.

W. ZENTMAYER.

Methods of Examination

IMPORTANCE OF ROENTGENOGRAPHY AND ERYTHROCYTE SEDIMENTATION RATE IN DIAGNOSIS AND TREATMENT OF DIFFERENT FORMS OF ARTICULAR RHEUMATISM ACCOMPANIED BY IRITIS. F. E. NOBOA-RECIO, Arch. de oftal. de Buenos Aires **16:** 36 (Jan.) 1941.

After stressing the occurrence of iritis and iridocyclitis of rheumatic origin and stating that recognition of the cause, which is often tuberculous or infectious (focal), is important therapeutically, the author claims that the ocular complication is milder in acute articular rheumatism than in the chronic forms. He classifies the chronic forms as progressive polyarthritis, ankylopoietic spondylitis and arthritis deformans. In the first two varieties there is an increase in the rate of sedimentation of the erythrocytes, which is absent in the third; differentiation of the first two types is possible by roentgen examination. In the three forms the iritis may be acute, subacute or chronic.

Two cases are reported. One patient was a woman 43 years old with chronic iridocyclitis with macular lesions, progressive polyarthritis and an increased sedimentation rate. The other was a man 42 years old with acute serous iritis, ankylopoietic sacroiliac spondylitis and an increased sedimentation rate.

The author recognizes that it is difficult to establish a relation between the ocular and the articular lesions.

C. E. FINLAY.

Neurology

NEUROLOGIC SYMPTOMS FOLLOWING EXTENSIVE OCCLUSION OF THE COMMON OR INTERNAL CAROTID ARTERY. A. B. KING and O. R. LANGWORTHY, Arch. Neurol. & Psychiat. **46:** 835 (Nov.) 1941.

Three clearcut cases of chronic massive occlusion of the carotid arteries on one side are reported. The causes of occlusion were infection, arteriosclerosis and embolus from an aneurysm. From consideration of the neurologic changes shown in these cases and study of the literature the authors arrive at the following summary:

"Occlusion of the carotid artery produces atrophy of the optic nerve and loss of vision in the eye on the same side, due to closure of the ophthalmic artery. The circulation is also impaired in the anterior choroidal, anterior cerebral and middle cerebral arteries, which are terminal branches of the internal carotid artery. There may occur softening of the brain, which is usually maximal in the field of the middle cerebral artery. Temporal hemianopia, due to involvement of the optic radiation, may be present in the visual field of the opposite eye. Contralateral hemiplegia and hemianesthesia, more marked in the face and arm than in the leg, also result; they appear to be due to cortical destruction rather than to injury to the internal capsule. If the left cortex is involved in a right-handed person, there may be a variable degree of aphasia. Dysarthria and dysphagia are often present during the first few days after the vascular occlusion, but disappear later."

R. IRVINE.

DIAGNOSIS AND MANAGEMENT OF SUBARACHNOID HEMORRHAGE. I. J. SANDS, Arch. Neurol. & Psychiat. **46**: 973 (Dec.) 1941.

This article is well worth reading for the discussion as to the pathogenesis of the findings, both clinical and laboratory, in subarachnoid hemorrhage, as well as for points in differential diagnosis for treatment.

To quote directly: "Hemorrhage into the subarachnoid space causes general, as well as focal, symptoms. The general symptoms are due to diffuse irritation and compression of the brain and to meningeal irrigation. The focal symptoms are due to irritation or disturbed function of certain parts of the nervous system. As the blood escapes into the subarachnoid space, intracranial pressure increases, causing headache, vomiting and dizziness. The headache may at first be localized to the nape of the neck, radiating upward toward the vertex, or the pain may be referred to the regions innervated by the fifth cranial nerve and presumably cause irritation of some or all of its branches. The pain is occasionally referred to either eye. The blood travels from the subarachnoid space along the sheaths of the optic nerves, causing blurring of the margins of the disks, and even papilledema. In the average case the eyegrounds present almost a pathognomonic picture; that is, the center of the disk appears more elevated than the periphery, and the margin is generally clearly outlined and the veins are not very tortuous, though they may be congested. The pressure in the optic sheath may cause obstruction of the central vein of the retina at a point where it leaves the optic nerve and enters the dural sheath, resulting in increased intraocular venous pressure and producing retinal hemorrhages. As the blood invades the sheaths of the other cranial nerves it produces disturbance of their functions. Thus, it may cause pupillary disorders, extraocular palsies, facial pareses, deviation of the tongue, and even of the lower jaw, ringing in the ears, difficulty in swallowing and respiratory and cardiac disturbances. The pupils may present a striking phenomenon—namely, marked variability in size, shape and reflexes from day to day. The pupils may be unequal in size; one or both may be irregular in outline, they may fail to react to light or in accommodation or they may show the paradoxic light reaction. . . .

"The syndrome of subarachnoid hemorrhage has frequently been mistaken for (1) septic meningitis, (2) sinusitis, (3) encephalitis, (4) tumor of the brain, and (5) uremia. The cervical rigidity, the Kernig sign, the pyrexia and the leukocytosis may lead to the diagnosis of septic meningitis. The headache, the vomiting, the photophobia and the elevation of temperature may suggest sinusitis. The drowsiness, the pupillary disorders and the involvement of the cranial nerves may simulate encephalitis. The headache, the changes in the eyegrounds, the convulsions, the vomiting and the unconsciousness may lead to the diagnosis of cerebral tumor. The loss of consciousness, the convulsions and the albuminuria may simulate a uremic condition. . . .

"Treatment is directed toward the relief of headache, the reduction of intracranial pressure, allayment of excitement, adequate feeding, consideration of surgical measures in suitable cases of aneurysmal rupture and rehabilitation of the patient."

Illustrative cases are reported.

R. IRVINE.

Ocular Muscles

FUNCTIONAL STRABISMUS. R. ARGAÑARAZ, Arch. de oftal. de Buenos Aires **16:** 409 (Aug.) 1941.

Argañaraz defines functional strabismus as a disease of the sensorimotor apparatus of vision characterized by a deviation of the visual axes. A disturbance of the motor function constitutes its initial symptom.

After describing in detail his views concerning normal binocular vision, the author states the opinion that the lesion in functional strabismus is toxic infectious, is transitory or permanent and is sudden in onset, appearing usually before the age of 3 years during convalescence from some infectious disease of childhood (measles, whooping cough, smallpox or gastrointestinal intoxication). The lesion, located either in the reflex motor center in the pons or in the cortical voluntary center, gives rise to a spasm of convergence, which if permanent is followed after some years by contracture and ankylosis of the tissues and aponeuroses over the internal portion of both globes.

The relation between accommodation and convergence in cases of a preexisting hypermetropia favors the occurrence of the spasm, acting, however, as an adjunct and not as a causative agent, as maintained by Donders. The presence of excessive hypermetropia, astigmatism and anisometropia in the squinting eye the author attributes to a persistence in it of fetal anatomic and physiologic characteristics, with often an amblyopia not accounted for by any coexisting ophthalmoscopic lesion.

Argañaraz considers the explanation that the squint is a congenital defect or the result of nonuse as erroneous; he considers it a sensorial hemiplegia with amblyopia or lack of vision in one eye. He likewise considers the explanation of the lack of diplopia in squint as the product of a psychic or cerebral suppression of the image of the squinting eye as another current mistake, the condition being due to an early interruption of the development of binocular vision.

Squint usually appears during the second and third years and often in the first; the earlier the onset, the worse the prognosis. When it occurs in the fourth or fifth year, the prognosis is better, owing to the development by that time of some degree of binocular vision and fusion. The deviation is apt to lessen and occasionally disappear in late infancy or childhood, and after 20 it is usually permanent.

In some cases there seems to be a familial predisposition.

Argañaraz does not accept Worth's explanation that the essential cause of strabismus is a defect in the fusion faculty. He considers the amblyopia the result and not the cause of the primitive deviation of the ocular axes.

Deviation of the eyes from other causes (corneal opacities, cataract, choroiditis, etc.) has no relation to functional squint. This is rarely cured by the ophthalmologist; in 6 to 9 per cent of cases parallelism is reestablished spontaneously; in 90 per cent the sensorimotor lesion is incurable.

The ocular defect is of first consideration. The prescription of glasses should be preceded by complete paralysis of accommodation for a week followed by full correction and constant use of the glasses. Good results

may be obtained in 8 to 10 per cent of the cases in which amblyopia and the disturbance of fusion are not pronounced.

The orthoptic treatment is the most scientific and rational, but its results in practice are poor, owing possibly to lack of patience and constancy. Its objective is to combat the amblyopia and favor the development of binocular vision and fusion by stereoscopic exercises. It should be commenced as early as possible. Of the different means employed, Argañaraz prefers Maddox' cheiroscope. Occlusion of the good eye for certain periods is an essential adjunct to this treatment.

Operative treatment for the correction of the deviation should not be undertaken until after the eighteenth or twentieth year. All surgical procedures give good results if indicated and properly executed. The author discusses in detail simple tenotomy, double tenotomy, tenotomy with advancement and double advancement.

C. E. FINLAY.

Operations

SURGICAL TREATMENT OF OCCLUSION OF THE PUPIL. J. HURTAULT and J. SVERDLICK, Arch. de oftal. de Buenos Aires **16:** 449 (Aug.) 1941.

The usual belief that in occlusion of the pupil the new-formed occluding membrane is adherent to the lens capsule is not always correct. After reference to a prior case of their own in which a cleavage was found between the membrane and the anterior lens capsule which permitted an extraction in toto of a complicating cataract, the authors report a case in which after a broad iridectomy separation of the iris from the lens was obtained by lateral introduction of a spatula under the pillars of the coloboma and was followed by extraction of the lens with a capsule forceps according to von Grósz's technic.

C. E. FINLAY.

Pharmacology

OPHTHALMOLOGIC STUDY OF DRUGS AFFECTING THE PERIPHERAL CIRCULATION: II. LOCAL EMPLOYMENT OF ACETYLCHOLINE. G. VON GROLMAN and E. ANGEL, Arch. de oftal. de Buenos Aires **16:** 283 (June) 1941.

Scientific reports on a favorable action of acetylcholine on visual field changes in glaucoma and optic neuritis determined the authors to investigate the action of this drug on the circulation of the eye.

Its pharmacology is first briefly reviewed. The drug is a vasodilator through parasympathetic stimulation. The effect when employed by subcutaneous, intramuscular or intravenous injection is evanescent. On account of rapid decomposition, intra-arterial injection is necessary. With this method the action is limited to the capillary zone of the injected vessel, as has been demonstrated in plethysmograms of different organs, the peripheral arterial pressure as a rule being diminished, with no increase or a slight increase in the systemic pressure.

Because of the failure of intravenous injection to affect the ocular circulation and the impracticability of employing the arterial route, the authors decided to study the effects of deep retrobulbar and subconjunctival injection, in 0.1 Gm. doses. The results on the intraocular

pressure in different ocular diseases are presented in tabular form. Of 31 cases, there was marked hypertension in 25, no change in 5 and hypotension in 1. Of 26 cases, tonoscopy revealed an increase in the systolic retinal arterial pressure in 25; in the diastolic pressure there was a slight increase in 20 cases, no change in 3 and a decrease in 3.

No change was noticed in the size of the pupil. No appreciable change was to be found in the caliber of the retinal vessels. There was pronounced dilatation of the conjunctival and of the cutaneous vessels. No variation was found in the visual field before and after the injection. A prior sympathectomy does not seem to have any effect on the action of the drug.

The paper is illustrated by abundant tabular reports, photographs and visual field charts.

C. E. FINLAY.

Refraction and Accommodation

A CASE OF TRANSITORY MYOPIA FOLLOWING RADIUM THERAPY. O. VON FIEANDT, Acta ophth. **19:** 11, 1941.

A 24 year old man received treatment for a squamous cell carcinoma on the limbus of his right cornea. Radium needles were applied for a period of four hours. In the eye, previously emmetropic, a myopia of 2.5 D. developed. This persisted for about a week. With the use of homatropine it was reduced by 0.5 D., returning to 2.5 D. after the effect of the homatropine wore off.

O. P. PERKINS.

Trachoma

ATYPICAL TRACHOMA. V. A. VICTORIA, Arch. de oftal. de Buenos Aires **16:** 440 (Aug.) 1941.

The author calls attention to the existence of attenuated forms of trachoma, especially in cities where conditions which exacerbate its malignity are lacking.

The symptoms are mild: itching of the lids, photophobia, sensation of a foreign body and some lacrimation. There is little or no secretion. Occasional nodules, visible biomicroscopically, are observed in the retrotarsal folds.

C. E. FINLAY.

Tumors

CONJUNCTIVAL PAPILLOMA. J. MALBRÁN and B. TOSI, Arch. de oftal. de Buenos Aires **16:** 243 (May) 1941.

Conjunctival papillomas vary according to the site of implantation, are composed essentially of a vascular connective tissue stem covered by thickened stratified epithelium and are not so benign as they are usually considered.

Seventy-five cases from the literature are tabulated. Clinical and histologic descriptions are presented of 2 cases in which the papilloma was in the tarsal conjunctiva and the semilunar fold, respectively. The authors recommend complete excision of the growth, supplemented when there is doubt of its epitheliomatous nature by radium therapy.

C. E. FINLAY.

Uvea

LATE DEVELOPMENTS IN A CASE OF GYRATE ATROPHY OF THE CHOROID AND RETINA (FUCHS). H. H. McGUIRE and W. P. McGUIRE, Am. J. Ophth. **24:** 657 (June) 1941.

A follow-up report on a case of gyrate atrophy of the choroid and retina first observed ten years ago is presented. The authors state:

"The case is reported again because of the unusual opportunity presented to study, over a period of 10 years, the slowly developing fundus changes, and for the purpose of exhibiting comparative illustrations of the early and late stages of the degenerative process.

"Subsequent observations have served to refute the opinion, expressed in the original paper, that the disease may be regarded as stationary.

"Night blindness, a symptom conspicuous by its absence in the early stages of the degeneration, is now definitely present."

W. S. REESE.

Therapeutics

ARSENICAL TREATMENT OF OCULAR HERPES ZOSTER. V. MUHLMANN and B. V. RE, Arch. de oftal. de Buenos Aires **16:** 434 (Aug.) 1941.

After calling attention to the relative frequency of herpes zoster, to the pain which often persists after the disappearance of the external lesions and to the duration of the condition from two to four weeks as a minimum, the authors call the disease a neurotropic ectodermosis produced by a filtrable virus which acts on the gasserian ganglion, or the root of the trigeminus nerve.

The causation is obscure; among the possible etiologic factors are metabolic disturbances (diabetes and gout), vitamin deficiencies, endogenous infections and intoxications (frequently syphilis), pulmonary tuberculosis and Pott's disease.

The authors report 4 cases in which arsenicals acted favorably and rapidly after other therapeutic agents had failed. C. E. FINLAY.

Society Transactions

EDITED BY DR. W. L. BENEDICT

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

KAUFMAN SCHLIVEK, M.D., *Chairman*

BRITTAIN PAYNE, M.D., *Secretary*

March 16, 1942

Case of Hypertelorism. DR. HENRY O. LITTLE (by invitation).

A boy 6 years of age with hypertelorism was first seen in the ophthalmic clinic of the Manhattan Eye, Ear and Throat Hospital in November 1939. His mother had a wide interpupillary measurement and a tendency toward hypertelorism. The vision was 20/200 in the right eye and 20/20 in the left and was not improved by correction. The sensorium was intact, and the mentality seemed unaffected. Neurologic tests gave negative results. Hypertelorism was suspected, and a definite diagnosis was made in October 1941 with the aid of roentgenologic studies.

DISCUSSION

DR. M. L. BERLINER: Two years ago Dr. Gartner and I (ARCH. OPHTH. **24**:691-697 [Oct.] 1940) were fortunate in observing a case of hypertelorism which might correctly be defined as a retention of the normal embryonic position of the orbits. I should like to show a few slides to demonstrate the anomaly in our case. As Dr. Little said, apparently this condition has been known for a long time, but in the recent literature most of the cases have been reported by pediatricians, and there have been only a few cases reported in the ophthalmic literature.

The first slide is a picture we found in a book on physiognomy by de la Porta published in 1855. He collected pictures of persons who had anomalies and compared them to animals. This picture probably represents the first recorded case of hypertelorism in which the author compared his subject's appearance to that of a cow; the slide demonstrates the so-called mongolian slant of the eyes, the broad nose and the bulging forehead. As Dr. Little said, the condition probably follows from an early ossification of the greater wing of the sphenoid bone and a consequent enlargement of the lesser wing, with the result that the frontal and the superior maxilla are pushed upward, outward and forward. This change necessitates the formation of the intranasal bone and in addition gives the nose a retroussé appearance.

The next slide is a picture of the patient on whom we reported, showing the typical features: the external canthus higher than the internal canthus, the low forehead and the wide distance between the

eyes. The patient had about 140 prism diopters of exotropia, and we attempted to correct some of it with only partial success. At the time of operation the internal rectus muscle was very atrophic. The first roentgen view shows the intranasal bone and the tremendous width between the bony orbital margins. The particular feature in our case was the presence of optic nerve atrophy in the right eye. In all previous cases published, this condition was not emphasized. If I understood correctly, Dr. Little said that there was amblyopia in one eye, and I wonder whether this might not have been due to pressure atrophy.

A roentgenogram of the left optic canal revealed it to be reduced to about 3 mm. in diameter. Normally it measures from 5 to 6 mm. In this eye the vision was normal.

The last slide shows the flattening of the right canal, measuring 2 by 4 mm., which we believe was the reason for the optic nerve atrophy in this case.

DR. TRACY J. PUTNAM: I should very much like to question whether the beaten silver appearance of the skull should be interpreted at this age as being evidence of increased intracranial pressure. It would have been interesting to have had a lumbar puncture with a pressure reading in Dr. Little's case, because it is conceivable that if there was an increased pressure, relief might have been afforded by some decompression operation, such as has been offered in some cases of papilledema in oxycephaly.

DR. HENRY O. LITTLE: Ophthalmoscopic examination failed to reveal any signs of optic nerve atrophy in either eye. The roentgenologic appearance of the right optic canal was normal, whereas the left canal seemed to be irregular in shape. I am persuaded, therefore, that the poor vision of the right was due to amblyopia exanopsia.

Case of Coats's Disease. DR. A. L. KORNZWEIG.

The patient was a white boy of 12 who complained of blurring of vision in the right eye one month before he was seen. His vision was 15/200 in the right eye and 20/20 in the left. The past history was significant because of a chronic cough of a spasmodic nature when the patient was between 2 and 5 years of age. The physical examination gave negative results except to reveal a pigmented nevus on the chest. Only the right eye was involved. The left was normal. The pathologic change in the right eye was limited chiefly to the retina. The smaller branches of several arteries and veins showed miliary aneurysmal dilatations. A massive exudate yellowish white in color was seen in the lower nasal field and in the distant temporal field. A grayish white exudate was present in the macular region and sprinkled around the disk, but the disk was not elevated. The condition fits the criteria for Coats's second classification, of massive retinal exudate with vascular changes. This case is of interest because the clear media permitted a careful examination of the retinal changes.

A painting and several photographs of the fundus were shown.

DISCUSSION

DR. HERMAN ELWYN: I want to discuss the pathogenesis of this disease. Coats described a number of cases and wrote two papers. In

his second paper he attempted to explain the disease; he attributed it to the presence of hemorrhage and expressed some opinion as to what caused the hemorrhage. He considered two causes: something abnormal in the blood and something abnormal in the small vessels. He believed that there was something wrong in the vessels but could not identify the trouble. Unfortunately, Leber, who had great influence, assumed that the disease was an inflammatory one and considered it as a necrotizing serous inflammation, and Duke-Elder still calls it an inflammation. When one looks at sections from an eye with Coats's disease and at reports and pictures, the criteria for inflammation are not there. There is no outpouring of leukocytes, and all one sees is a hemorrhage in the retina which, because it is so large, cannot be absorbed and therefore becomes encapsulated. The question then remains. What causes the hemorrhage? In practically every case that has been reported, sections have shown malformed vessels in one portion of the retina. Such vessels are dilated, form dissecting aneurysms, frequently are ruptured and have malformed walls. They looked like a cavernous angioma in 1 of Coats's cases. The blood flow in such vessels is apt to be sluggish, stasis results and the vessels rupture and bleed. This sequence is characteristic of telangiectasia, which, I believe, is the primary condition. The circulation of the eye is part of the cerebral circulation, and the lesions of telangiectasia are analogous to those found in the pons and in other parts of the brain as isolated deformities. Primarily, a mesenchymal deficiency due to unknown factors, causing malformed vessels with repeated hemorrhages, makes up Coats's disease. It is rather peculiar that Leber, who reported in 1912 such a case as Dr. Kornzweig's, in which there were miliary aneurysms, a form of Coats's disease, was inclined to the causation of inflammation in this disease. The condition is localized telangiectasia with secondary changes.

DR. SIGMUND A. AGATSTON: I hope this is a case of Coats's disease. I have never been able to find out exactly what "Coats's disease" means. It is described as the presence of massive exudate, a tendency to hemorrhage and proliferation. However, no one has really given an exact conception of the cause of the rupture of the vessels; whether it is due to tubercles of veins or arteries or to some other pathologic change has never been certain. I looked at Dr. Kornzweig's patient, and my impression was that his case was similar to the one which Dr. Minsky reported. I am not so sure that the condition was Coats's disease. I should like to think it was. However, some years ago there were a number of similar cases at Bellevue Hospital, and 2 were presented before this society about ten years ago. At that time I observed 9 cases at Bellevue Hospital in which the ocular disease came on with loss of vision during an attack of influenza and the fundus picture showed an optic neuritis. This stage was followed by a recession of the optic neuritis and gradual return of vision, with the appearance of scattered white spots, just as in Dr. Kornzweig's case, and with a star-shaped figure around the macula. In all these cases the vision returned almost to normal, and the white spots that were seen during the time of recession of the optic neuritis gradually disappeared. While Dr. Kornzweig's case may be an instance of Coats's disease, it reminded me of that group of cases, particularly as Dr. Kornzweig stated that

the loss of vision started rather suddenly and the vision seemed to be returning gradually. So, while I do not want to throw cold water on this diagnosis, I think the possibility of the other condition ought to be kept in mind.

DR. A. L. KORNZWEIG: The conception of Coats's disease, especially in its early stages, is a difficult one to understand. Not many cases have been observed in the early stages; most have been observed in the late, or advanced, stages, with massive exudate, and the initial pathologic change was not clear. I considered this to be a case of Coats's disease because there was massive exudate with vascular changes in one eye in a young man, which is just what Coats described in his two papers.

Case of Nevus Verrucosus of the Scalp with Involvement of the Conjunctiva. DR. A. RUSSELL SHERMAN.

This article will be published in full in a later issue of the ARCHIVES.

Multiple Sclerosis as an Etiologic Factor in Retrobulbar Neuritis. DR. WILLIAM L. BENEDICT, Rochester, Minn. (by invitation).

This article will be published in full in a later issue of the ARCHIVES.

Eye Dominance: Its Nature and Treatment. DR. IRA WILE.

This article will be published in full in a later issue of the ARCHIVES.

Book Reviews

Visual Mechanisms. Biological Symposia, Volume VII. Edited by Heinrich Klüver. Price, $3.25. Pp. 322. Lancaster, Pa.: The Jaques Cattell Press.

This volume is an outgrowth of the celebration of the fiftieth anniversary of the University of Chicago in 1941. As a part of that celebration eight papers dealing with visual mechanisms were presented; under the editorship of Dr. Heinrich Klüver, professor of experimental psychology at the University of Chicago, four papers have been added. The additions have been well selected and constitute much of the most interesting material in the volume.

This book makes its appeal to the medical biologist. It is written chiefly from the biophysical, biochemical and neurophysiologic points of view. With fourteen authors in widely differing fields contributing, a smoothly integrated progression from the physical to the psychologic could hardly be expected, and yet this has in a large measure been achieved.

Most assuredly this is a book for the ophthalmologist to read. He will not understand a great part of it, but he will, on completing it, have enlivened his thinking and enjoyed a personally conducted tour into twelve new frontiers in research dealing with his specialty.

The editor in his foreword expressed the situation neatly after referring to the satisfaction Charles Darwin derived from reading articles which (according to himself) he could not understand. Klüver exhorts the specialist in problems of vision to seize on every opportunity to expose himself to facts and hypotheses not germane to his own field.

In organization the symposium begins with papers on biophysics and biochemistry, proceeds to neurophysiologic studies, digresses to neuroanatomic and retinoanatomic matters and concludes with cerebral and psychologic considerations. The volume furnishes ideal material for reflective reading.

Prof. Selig Hecht, of the laboratory of biophysics, Columbia University, contributes the first chapter, which presents physical data on a threshold stimulus-response situation, and concludes that the minimal visual response may be achieved when five to fourteen retinal rods have absorbed one quantum each of maximally active visible radiations.

Arlington Krause, of the University of Chicago, continues with a study of the chemical structure of vitamin A and related compounds and presents a list of diseases which cause retinal avitaminosis A.

This is followed by one of the papers added to the original symposium. George Wald, of the biological laboratories, Harvard University, discusses the visual systems and vitamin A. No part of the symposium excels this fascinating presentation. Wald's great gift for profitable exploration is supplemented by a rarely encountered literary style. He reveals the progress of his studies with such logic and clarity of expression that the reader recaptures much of the pleasure he had in discovering Henri Fabre.

The fourth paper deals with anoxia in relation to the visual system. E. Gelhorn, of the departments of physiology and psychiatry, University

of Illinois, presents interesting data relating blood oxygen and sugar levels to the functioning of the cerebral visual system. The paper is highly technical.

S. Howard Bartley, of the laboratory of neurophysiology, Washington University School of Medicine, St. Louis, presents a small portion of the great amount of work he has done in electrophysiology. His contribution to this volume, entitled "Visual Sensation and Its Dependence on the Neurophysiology of the Optic Pathway," is an elaboration of the paper read at the original symposium under the title "Some Correlations Between the Sensory and Neurophysiologic Aspects of Vision." Some of the newer concepts of vector and field hypotheses are touched on; more are to be found in his volume on "Vision," published in 1941. However controversial the subjects of vector and field analyses as they relate to visual functions, Bartley is among those who are bringing to the front of visual research the tools of electrophysiology.

The sixth paper is a short but lucid and well presented exposition of the "Alpha Waves in Relation to the Structures Involved in Vision," by Theodore Case, of the division of psychiatry, University of Chicago. In this highly technical presentation of 6 cases, in all of which the lesions were determined by operation, Dr. Case has greatly facilitated understanding of his discussion by clearcut drawings of the field defects, the location of the lesion in the optic pathway, the topographic location in the brain, the position of the electrodes and the resultant alpha wave patterns. If more case reports were as well illustrated, there would result a great deal of improvement in understanding with an enormous economy of words. So expressive are the author's graphic presentations that this and similar work (such as that of Marshall and Talbot, which follows) leads one to speculate on the future of perimetry. It is conceivable, although at present far from achievement, that the electroencephalograph may take the place of the perimeter. Certainly now in cases of intracranial lesions they must supplement each other.

W. H. Marshall and S. A. Talbot, of the physiologic optics laboratory, Wilmer Ophthalmological Institute, Johns Hopkins University, present one of the longest and most complicated papers of the symposium, entitled "Recent Evidence for Neural Mechanisms in Vision Leading to a General Theory of Sensory Acuity." Essentially they discuss ways in which discrimination data may be interpreted, but the discussions and interpretations are so abstruse that one proceeds under a constant awareness of insecurity. This chapter should be required reading for any ophthalmologist (or optometrist) who naively believes that the measurement of visual acuity is a simple matter encompassed by a notation of 20/20, 14/14, etc. "Neural amplification," "border inhibition," "accentuation of gradients," "control of thresholds," "lability of pathways" and "synaptic aggregates" are all phrases which, with "neural saturation," "equilibration," "facilitation" and "occlusion," will require clearer understanding and more lucid expression before being incorporated into a generally accepted theory of vision.

While Marshall and Talbot have achieved a high degree of accuracy (greater than that achieved with perimetry) in localizing the representation of the retinal areas in the occipital lobe, they do not present this work in the present discussion. There is presented, however, in the eighth paper of the symposium, "The Functional Organization of the Occipital Lobe," by von Bonin, Garol and McCulloch, an excellent

account of a careful reexamination of the functional organization of areas 17, 18 and 19 (Brodman) in the occipital cortex of the primate. Their experiments were carried out on 12 macaques and 4 chimpanzees and resulted in the proposal that the three functionally distinct areas they localized shall be called the striate, parastriate and occipital areas.

The ninth paper of the symposium, "Anatomy of the Retina," by S. L. Polyak, University of Chicago, consists of eight pages of histology with one plate, a condensation to about 1 per cent of his recently published (1941) exhaustive volume on "The Retina," which contains six hundred and seven pages, with one hundred plates. Since the abstract was made by the author, it should furnish excellent material for a quick review of retinal histology.

In the tenth paper Gordon Walls, of the ophthalmic research laboratory, Wayne University, Detroit, under the title "Visual Cells and Their History," gives a fascinating account of the natural history and differentiation of the visual cells. This paper is profusely and beautifully illustrated. Walls has a straightforward, clear and conversational style of writing which makes reading a pleasure in spite of the disconcertingly encyclopedic nature of his material. Unless one has a wide knowledge of comparative embryology and anatomy, one is perforce required to accept on faith many statements given by the author as interesting facts. It is to be hoped that the author is a sure guide. He obligingly furnishes the reader with a double page folded insert showing a "tentative schema of the evolution of the visual cells in vertebrates," and with this map to guide one and refreshing comments in the text, one can spend from one to a hundred delightful hours under his ciceronage. His skepticims are as refreshing as his faiths, and both contribute to the interest of the article. Without intent to disparage the technicalities and abstrusities of other chapters, it may be said that the change of mental pace in this paper is a happy relief. One, in effect, relaxes from frowningly close and critical observation of finely knit thinking and walks about a bit in the open with endless vistas of historical evolution to contemplate. The reader ends the chapter with a faint regret that more gross notes could not have been included—particularly some more of the story of the evolution of form and function of the eye, its vascularization, etc. Some of these notes will be found in the author's "Vertebrate Eye," published in 1942.

The editor of the symposium, Dr. Heinrich Klüver, contributes in the eleventh paper, "Functional Significance of the Geniculo Striate System," a study of behavior of macaque monkeys after bilateral destruction of the striate cortex. In such animals degeneration of the lateral geniculate bodies follows, and hence effects mediated by these bodies can be studied before and after functional isolation.

Klüver's paper is one of the longer analytic studies in the symposium. In it definite technical procedures are described or referred to and the effects of such procedures analyzed. A hope is expressed that through the utilization of correlated surgical, electrophysiologic and behavior technics a greater understanding of what happens in the cerebral nervous system during visual functioning will be achieved.

The symposium closes properly with a psychologic chapter on "The Problem of Cerebral Localization in Vision," by Prof. K. S. Lashley, of Harvard University. Professor Lashley discusses the vexing questions of stimulus equivalence and motor equivalence and quickly evaluates

the probabilities of their elucidation. The field theories and vector theories do not receive much support from his brief discussion. He looks with greater hope toward an explanation based on some form of interference pattern. The chapter is an excellent ending to the volume, since it leaves with the reader much material for speculative discussion as he departs, enriched, from the symposium. LEGRAND H. HARDY.

A New Classification of the Motor Anomalies of the Eye Based Upon Physiological Principles Together with Their Symptoms, Diagnosis and Treatment. By Alexander Duane, M.D. Lithographic reproduction of a brochure published in 1897. Price, $2. New York: Dr. James W. White, 15 Park Avenue.

In this essay Dr. Duane brought forth his classification of motor anomalies and outlined the symptoms, diagnosis and treatment of the various types of deviation. The present day widespread acceptance of his teachings is proof of the soundness of his views. It is refreshing and stimulating to reread this brochure in its original form and to ponder over how little escaped this master mind. The intervening forty-six years have brought but few changes, and these few have been refinements in operative procedures, etc., with the basic principles remaining unchanged. The beginner in ophthalmology can find nowhere else such a clear exposition of the diagnosis and treatment of the motor anomalies. While this valuable monograph has been freely quoted many times by many authors, the original issue of it has been out of print for a long time. Ophthalmology, therefore, is deeply indebted to Dr. White for republishing it. J. H. DUNNINGTON.

Manual on the Use of a Standard Classification of Causes of Blindness. Prepared by C. Edith Kerby for the Committee on Statistics of the Blind. Complimentary. Pp. 25. New York: American Foundation for the Blind and National Society for the Prevention of Blindness, 1940.

This concise but detailed guide has for its purpose the collection, recording and interpretation of data on a uniform and scientific basis. Stress is laid on the importance of ocular examinations by ophthalmologists, on study sufficient to determine the underlying causation whenever possible, on detailed registration in standardized form and on authoritative interpretation of the summarized findings.

The collection of data in accordance with these principles should be of value not only in statistics but, eventually, in the prevention of blindness and the conservation of vision. PERCY FRIDENBERG.

CORRECTION

In the article by Dr. Henry Minsky entitled "Concept of a Zonular Chamber: Preliminary Report," in the August issue (ARCH. OPHTH. **28:** 214, 1942), an error appears in the second sentence of the legend under the illustration, which should read "The hyalozonular leaf" instead of "the posterior zonular leaf."